THE JOURNAL OF PERIODONTOLOGY

January
1955

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY
VOLUME 26
NUMBER 1

RK 1
J855
v.26.

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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

Thirty-ninth Annual Meeting
THE AMERICAN ACADEMY OF PERIODONTOLOGY
San Francisco, California

Spring Seminar
May 2, 3, 4, 1955
Ann Arbor, Michigan

A126652

DR. HAROLD RAY

President

American Academy of Periodontology

1954-1955

Dr. Harold Ray of Palo Alto, California, was installed as President of the American Academy of Periodontology at the 38th Annual Meeting of the Academy of Miami. Unfortunately, Dr. Ray was unable to be present because of ill health, but his interest in periodontology and services to the Academy warranted honoring him with the position as President.

Dr. Ray was graduated from Northwestern University College of Dentistry in 1926 and taught on the staff there in dental pathology until 1933. In 1949 he was given a Master of Science degree from the University of Illinois and is now Associate Professor of Periodontology at the University of California College of Dentistry. He is also Chairman of the Division of Periodontology in that institution.

Dr. Ray is well known for his writing in the field of periodontology and his leadership in the activities of the Academy.

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FIFTY-FIVE

DYNAMIC EQUILIBRIA OF ORAL TISSUES

I. Theoretical Considerations*

by PHILIP PERSON, D.D.S., PH.D., *Washington, D. C.*

IN most intact living tissues, dynamic, overall equilibria are established, known as the "steady state." These are not the equilibria of classical chemical thermodynamics. The two fundamentally different equilibrium systems may seem to be the same outwardly because neither appears to be changing with time. Thermodynamic equilibria, however, are *reversible* processes, and for a given sequence of reactions to be in overall equilibrium, *each* of the individual reactions involved *must* also be in equilibrium. In a series of biological processes in the steady state, on the other hand, in addition to reversible reactions, *irreversible* reactions *may* be involved, and it is *not necessary* that any of the individual reactions be in chemical equilibrium (although they may be).[1] As we shall see, the steady state concept, and that of "the dynamic equilibria of body constituents"[2] provide real and stimulating perspectives for analysis of metabolic problems. The concept of the "dynamic equilibria of body constituents" implies that there is a continuous flux and interchange of building and energy yielding materials between various tissues and body compartments.[2,3] This exchange serves to preserve the morphologic and biochemical integrity of tissues and organs to such an extent that although they appear outwardly to be unchanging, they are in fact undergoing unceasing change. This is the miracle, and thus far, an untouched and unsolved central problem of biology. The adequacy of this concept has been established during the last fifteen years by the work of Schöenheimer,[2] Allison,[3] Whipple,[4] and others.[5,6]

The steady state, and the dynamic equilibria of bodily constituents, are made possible by intracellular protein catalysts known as enzymes, mediators of the metabolic reactions essential to life. For many years, enzymes were considered to be catalysts in the classical, or "Ostwald" sense, i.e., they did not initiate a reaction or make it possible; they merely increased the speed or rate of the reaction, without entering into it or experiencing any chemical change. Today, it is known that this definition of catalysts does not apply to enzymes. These proteins *do* initiate biochemical processes which otherwise would never occur, and they *do* participate in the reactions they accelerate.[7] The in vitro oxidation

*First of a series of three articles. From the Department of Dental Research, Army Medical Service Graduate School, Walter Reed Army Medical Center, Washington 12, D. C.

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of glucose to carbon dioxide and water by acid at high temperature is not the same process as the biological, enzymatic oxidation of glucose to carbon dioxide and water within the cell. The over-all energy yield is the same by either method, but the intermediates and path followed in the cell cannot be duplicated by in vitro chemical oxidation. The work of the enzyme is to make possible, under conditions compatible with life, the biochemical and biophysical processes essential to life.

DEFINITIONS AND CONCEPTS

1. Metabolism—In this series of papers, the word *metabolism* embraces the chemical and physical changes experienced by substances entering into and acted upon by living cells and organs. *Intermediary metabolism* is the occurrence of these changes at cellular and subcellular levels, with particular emphasis upon unit processes and intermediates.

2. Biochemical lesion—Often the initial disturbances in a disease state are biochemical in nature, with subsequent morphological changes and clinical symptoms developing from a basic "biochemical lesion."[8] One must acknowledge, however, that at the sub-cellular macromolecular levels on which life processes occur, morphology and biochemistry may be inseparable.[9-12]

3. Energy metabolism—Since most metabolic reactions are enzymatic, and since tissues require adequate supply and utilization of energy, a brief paragraph on this aspect of metabolism is in order. Life processes such as synthesis of protein, muscular contraction, secretion, and transport of ions and molecules against concentration gradients, etc., are mainly endergonic; that is, they will occur only if energy is supplied to the cellular systems in which they operate. In mammalian tissues, as well as those of most other animals, the main source of such energy is the oxidation of carbohydrate. The overall picture is that of the enzymatic splitting of large, relatively complex, organic molecules into smaller units, with simultaneous release of stored covalent bond energies originally derived from the sun's quanta. The liberated energy is channeled into related systems for utilization, or storage in other chemical bondings. At cellular and sub-cellular levels, these reactions are dependent upon macromolecular and cytoarchitectural factors which permit delicately poised regulation and integration.

4. Protein metabolism—In essence, protein metabolism is "the story of amino acids singly, and in various combinations, as they enter into and become part of the chemical matrix of living systems."[13] There is a living chemical matrix of protein in which centers form where the dynamic equilibria of life develop. This living chemical matrix of protein metabolizes, and in turn, is metabolized. Since there are no protein reserves in the sense that there are carbohydrate or fat reserves, the living proteins of the intestinal tract, liver, oral tissues, skeleton, etc., serve as the "protein stores" of the body, and are utilized, together with dietary proteins, to maintain the nitrogen integrity of the body. Tissue and plasma proteins and amino acids and those of dietary origin form a system in dynamic equilibrium, the "protein metabolic pool," as they are absorbed from the intestine. Dietary protein derivatives, as they enter this system from the intestinal tract, intermingle with and become indistinguishable from those of the tissues. Different tissues contribute nitrogen to, and withdraw nitrogen at varying rates from, the metabolic pool in accord with metabolic need and regulating influences. There are instances when given tissues may lose protein to supply demands elsewhere in the body. Thus in protein depletion, protein from the cytoplasm of intestine and liver is lost so that the integrity of plasma proteins may be maintained. Approximately 30 times as much body nitrogen is lost from the soft tissues, as from the circulating proteins during protein depletion.[4] When a tissue loses protein to supply a demand elsewhere in the body, the lost nitrogen often

represents active, metabolizing protein, including enzymatic protein.[14]

ORAL TISSUES AND THE METABOLIC POOL

It has long been recognized that oral tissues participate in the metabolic equilibria discussed previously. So much is this so, and so significant is this participation (in addition to the purely dental interests involved) that it has been said, "Physicians interested in metabolic and nutritional disturbances now examine the mouth before they take a blood pressure reading. A summary of the nutrition surveys conducted here and abroad reveals that over half the examination is conducted in and around the mouth."[15] The changes occurring within oral tissues are not only of importance to the dentist, but an understanding of the changes in terms of the fundamental biochemical and biophysical processes involved will be of tremendous value to all who are interested in nutrition and metabolism, and disorders thereof.

In any oral disease, mechanical, nutritional, microbiological, endocrine, nervous, and genetic factors are interwoven. Notwithstanding this, and whether the etiologic factors are predominantly "local" or "systemic," the net result is a change in the *metabolism* of the affected tissues. Clinical symptoms are but reflections of this fact.

From the mass of case reports, articles, and reviews in the literature of oral tissue change in health and disease, two observations emerge to challenge us within the context of this discussion. Keeping in mind our previous reference to the complexity of etiologic factors involved in oral tissue disease, the first observation is that oral tissue metabolism is altered in many generalized, nutritional, and metabolic diseases, and that these alterations are reflections of changes occurring in the dynamic metabolic equilibria existing between oral tissues and the various other tissues and compartments of the body. The second observation is that oral tissue change and pathological condition, often identical in character and course to those mentioned above, may be observed in individuals in whom there is absence of any detectable metabolic or nutritional disturbances.

An example of such a paradox is the condition known as periodontosis (diffuse alveolar atrophy, paradentosis, etc.), a syndrome which is one of the borderland territories of dentistry; recognized by many, treated by few, and understood by none. Cited below, very briefly, is an interesting clinical case in which the nature of this paradox becomes evident.

A 22-year-old married Negress was admitted with complaint of difficulty in chewing because of pain resulting from eating hard foodstuffs. Examination of the mouth revealed marked mobility of the teeth and some bleeding from the gingiva. Roentgenograms revealed severe generalized alveolar bone loss. The degree of tooth mobility and the marked bone loss could not be attributed to poor oral hygiene, malocclusion, or any local factors. The other physical findings and history were negative.

The laboratory findings included:

Hemoglobin—12 gms
Red Cell Count—4.1 million
White Cell Count—6950
Sedimentation Rate—20mms/hr
Urine—Negative
Wassermann—Negative
Cholesterol—320 mgm % (upper limit of normal range with method used)
Alkaline-Phosphatase—9 King-Armstrong units.

The patient was hospitalized and the calcium metabolism studied by means of the Bauer-Aub diet. The calcium excretion over a 3-day period was normal. Blood calcium and phosphorus values were also normal (i.e., 10.6 and 3.6 mg per 100 cc. respectively). Roentgenograms of the pelvis and lumbar vertebrae disclosed no abnormalities.

Consideration of this case reveals the

occurrence of a rapid alveolar bone loss in the absence of detectable metabolic disturbances. On the other hand, one sees a similar oral picture in cases of unquestioned metabolic abnormality, i.e., in certain cases of diabetes mellitus. Consideration of so-called local factors of oral hygiene or occlusion reveals that they are insufficient to explain the nature and rate of alveolar bone loss, in either instance. As might be expected, the rate of bone loss may be accelerated in certain diabetics, but this is not necessarily so in all cases. It would be wonderfully simple to make out an explanation for the events in the diabetic patient, based upon known derangements in carbohydrate, protein, and mineral metabolism associated with the syndrome. At times, even though the patient is being controlled with insulin, one encounters a rapid alveolar bone loss. It might be argued that even the best regulated diabetics must experience episodes in which insulin levels are either too low or too high, with resultant homeostatic disturbances. Over a period of time, the effects of such non-physiologic conditions might be cumulative, and thus result in alveolar bone loss, among other things. Even if such an argument were admissible, it would fall flat as an adequate explanation when confronted with the fact that it obviously could not be used in the non-diabetic.

There are many other oral tissue changes, some frankly pathological, others not, which might be used as examples of the paradox of the clinical manifestations of apparent metabolic disturbances. The general practitioner, orthodontist, oral surgeon, endodontist, pedodontist, as well as our medical associates, all meet with instances each day, in which disturbances of oral tissue metabolism are reflected as clinical symptoms and patient's complaints, inexplicable with our present knowledge.

We may consider also the numerous oral tissue changes associated with frank nutritional deficiency. Again one faces the paradox of the existence of similar, and often identical changes in patients in whom nutritional deficiency or interferences in absorption of nutriments cannot be demonstrated.

Surely much of the answers to these problems lies in a knowledge of the basic metabolic processes occurring within oral tissues themselves, and of the dynamic equilibria by which these processes are related to the economy of the organism.

We will not formulate at this time attractive diagrammatic schemes of proposed interrelationships, although hypotheses do exist which would serve this purpose. Such formulations would inevitably be based upon experimental data from studies of intermediary metabolism involving other than oral tissues, since data for the latter do not exist at the present time.

It is our belief that in the next decade sufficient data pertaining to oral tissue metabolism in terms of basic biophysical and biochemical processes will have been accumulated to permit the formulation of hypotheses concerning the nature of, and the mechanisms involved in the dynamic equilibria of oral tissues. The two papers which follow in this series are a beginning designed to obtain some of the data which will be needed "for the grace of the greater knowledge we seek."

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A Sex Difference in Epithelial Cells of Human Gingiva

by A. S. MARWAH, B.D.S., AND J. P. WEINMANN, M.D., *Chicago, Illinois*

UNTIL recently it was thought that the sex difference in the chromosomal equipment of mammals might become visible during mitosis but could not be observed during the interphase, when the chromosomes are fragmented and the chromatin diffuse. Interphase differences in mammalian nuclei of the two sexes were first described for nerve cells in the cat[1] and have since been noted in the neurons of dogs, minks, martens, ferrets, raccoons, goats, skunks,[5] and monkeys,[7] and also for a variety of other tissues in the cat, including many types of epithelial cells, cartilage, endothelium and smooth and striated muscle.[4]

When a difference can be recognized in mammalian cells, it is seen to consist in an excess of chromatin in the female nucleus. This excess of substance, which stains with basic dyes and gives positive tests for desoxyribose nucleic acid, is visible as a discrete particle, to which the name "sex chromatin" has been given. This is larger than other chromatin particles in the nuclear space, stains more deeply, and in the majority of instances lies adjacent to the nuclear membrane and follows its outline. It is most clearly visible when a nucleus is cut in the plane of its largest diameter. In sections of 5 microns thickness, it can be made out in about two-thirds of the cells. A similar, but much smaller particle of chromatin can be made out in a low percentage of the corresponding cells of male animals. Its smaller size and the associated low incidence of recognition in the male are ascribed to the fact that the particles in question are derivatives of the sex chromosomes, which in mammals are paired in the female and occur singly or with a diminutive second chromosome in the male.

Studies on man have been limited to the nervous system, the neutrophil leucocytes, and the epidermis.[2, 6, 3] For nerve cells, the recognition of a sex difference was limited to some few locations, and elsewhere was difficult. In the Malpighian layers of the skin, however, a clear cut difference could be made out.

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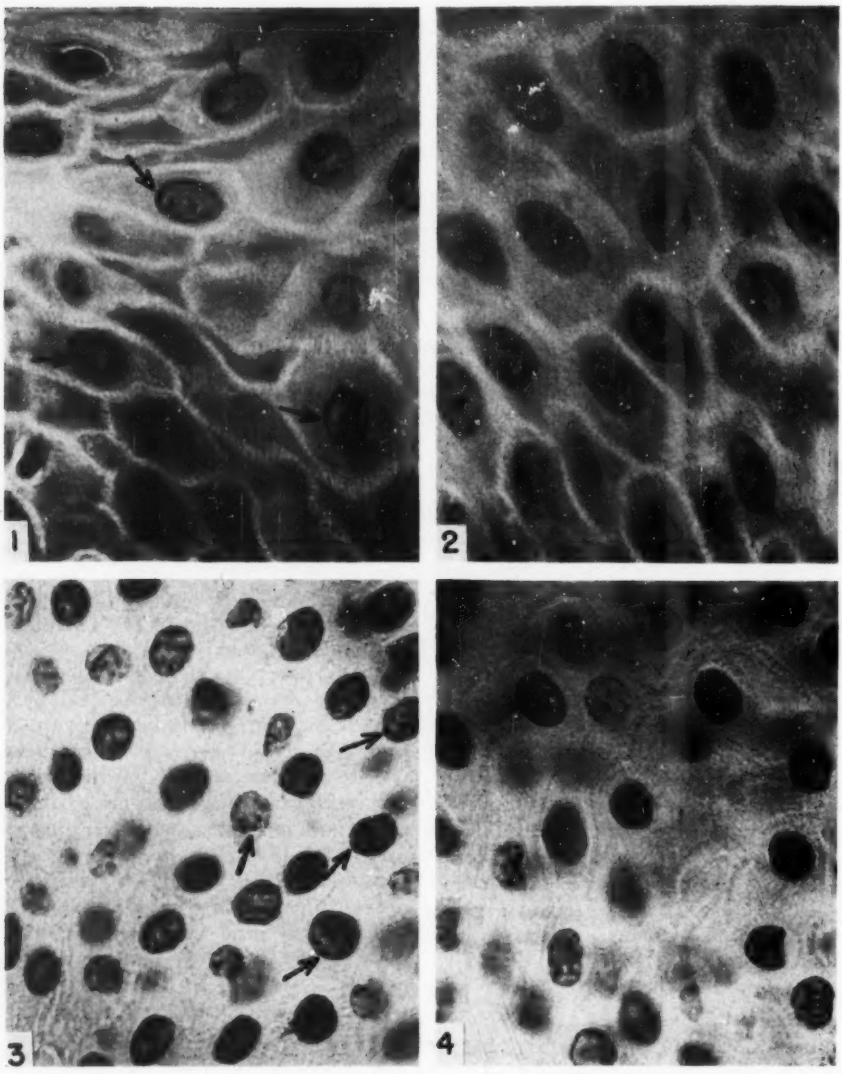
This investigation was carried out under Contract W-49-007 MD 496 with the Medical Research and Development Board, Office of the Surgeon General of the United States Army.



Figs. 1 to 4. Prickle cells of human gingiva. (Magnification, X1200).
Fig. 1. Female. Note the "sex chromatin" adjacent to nuclear membrane indicated by arrows. (Hematoxylin-eosin stain).
Fig. 2. Male. Note the absence of "sex chromatin." (Hematoxylin-eosin stain).
Fig. 3. Female. Note the "sex chromatin" indicated by arrows. (Feulgen-stain).
Fig. 4. Male. Note the absence of "sex chromatin." (Feulgen-stain).

The present study has extended similar findings to the epithelial cells of the human gingiva.

Material and Methods: Most of the material examined for this study were the routine sections of human biopsies which

are used for diagnosis. Biopsies from 81 patients whose gingivae appeared clinically normal and who ranged in age from 4 to 78 years were examined under oil immersion. These tissues had been fixed in Zenker formalin, cut at 5 to 7 microns and stained with hematoxylin-eosin. Eight additional cases were specially prepared for Feulgen staining according to the technique described by Stowell.[8] These were cut at 5 microns.

Findings: There is a clear cut sex difference in the epithelial nuclei of human gingiva. Even in the routine biopsy sections and without previous experience, sex was diagnosed correctly in 78 of the 81 cases. No mistake was made in diagnosing the 46 male cases, which goes to show that the other chromatin particles in the nucleus are not readily mistaken for the sex-dependent particle.

Gingival nuclei in the female (Figures 1 and 3): As in the human epidermal epithelium, the sex-dependent difference consisted in the presence of a large chromatin particle in the nuclei of female specimens. The largest diameter of this particle measured about 1 micron. The particle was usually situated just inside the nuclear membrane, following its outline. On the side away from the membrane, it was rounded, straight or irregular in outline. In hematoxylin-stained sections, the particle was more deeply stained than the nucleolus. In Feulgen-stained sections, it stained more deeply than the other Feulgen-positive chromosome fragments.

A count of the cells in which the nucleus contained a large, Feulgen-positive particle amounted to about three-fourths of the cells. For cells in which such a particle was adjacent to the nuclear membrane the count amounted to 50 per cent. The spatial orientation of the particle appeared to be random with respect to the basement membrane. It was more difficult to identify in the cells of the basal layer than in those of the spinous layer.

Gingival nuclei in the male: A smaller, similarly located particle could be made out in one or two per cent of the nuclei in the Feulgen-stained sections. In the hematoxylin sections, it could not be identified with certainty (Figures 2 and 4).

SUMMARY

Human gingival epithelium was found to be similar to human epidermal epithelium in showing a distinct sex difference. This difference consisted in the presence, in three-fourths of the nuclei of female specimens, of a large Feulgen-positive particle, usually adjacent to the nuclear membrane. Nuclei of male specimens showed a similar smaller particle in from one to two per cent of the cells.

Acknowledgments: We wish to express our appreciation to Dr. B. Orban, for permission to use his biopsy material for this study, and to Mr. W. Winn for his contribution of the microphotographs.

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Role of Trauma from Occlusion in Initiation of Periodontal Pocket Formation in Experimental Animals

by IRVING GLICKMAN* AND LEON A. WEISS,** *Boston, Massachusetts*

THE frequency with which trauma from occlusion† and periodontal pockets occur as associated clinical findings could conceivably lead to the assumption that trauma is a factor in the initiation of periodontal pocket formation. Microscopic studies of human autopsy material are abundantly documented with the injurious effects of excessive functional forces upon the tissues of the periodontium.[1] Depending upon the severity of the force, the changes vary from a simple widening of the periodontal membrane associated with an increase in normal osteoclastic activity, to compression of the periodontal membrane with hemorrhage and thrombosis, degeneration and necrosis of the periodontal membrane, excessive resorption and resorption of the tooth surface.

The specific question as to whether trauma from occlusion initiates periodontal pocket formation has not been explored to any great extent. Some observations in human autopsy material indicate that periodontal pockets are deeper in relation to teeth subjected to excessive occlusal stresses,[2, 3] while others afford no evidence that overstress alone can cause a deepening of the periodontal pocket.[4] The few studies in which the problem of initiation of periodontal pockets was dealt with experimentally led to difference of opinion regarding the exact role of trauma.[5, 6]

The question of whether trauma from occlusion can actually initiate pocket formation is a basic one in clinical periodontology which warrants further investigation. The validity of the assumption of a cause and effect relationship between trauma from occlusion and the initiation of pocket formation based upon clinical observations is of necessity subject to the qualifying limitations imposed by the difficulty of carefully controlling such observations. Nor do autopsy findings lend themselves to definitive conclusions regarding cause and effect relationships. Animal experimentation was therefore selected as the most promising method of approaching this problem.

EXPERIMENTAL PROCEDURE

Nine dogs weighing from 21 to 34 pounds were used in this study. Six served as experimental animals, three were used as controls. To induce excessive force upon the periodontium a cast metal onlay was cemented on the maxillary anterior teeth. The onlay was designed so that it occluded with the mandibular teeth in an edge to edge relationship and forces were transmitted in the long axis of the teeth. With the onlays in position, complete closure of the jaws was prevented, and functional contact of the natural dentition was not possible. In three of the experimental animals, the onlay was cemented on the six maxillary anterior teeth (Fig. 1). In the other three experimental animals, only the three maxillary anterior teeth on the left side were covered with the onlay (Fig. 2).

The animals were sacrificed after the onlays had been in position for intervals of from three to one hundred and ten days. The jaws were removed, fixed in 10 per cent formalin, decalcified by the formic acid-sodium citrate technique,[7] and embedded in celloidin. Serial mesio-distal sections were cut and stained with either hematoxylin and eosin or Mallory's aniline blue stain.

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†"Trauma from occlusion" is used instead of "Traumatic occlusion" because of the somewhat inaccurate implications of the literal interpretation of the latter term.

Fig. 1. Radiograph showing onlay in position on six maxillary anterior teeth.

MICROSCOPIC FINDINGS

Periodontal Membrane: In general, the periodontal membrane of the maxillary anterior teeth in the experimental animals was wider than in the controls and presented an altered microscopic appearance. In addition, in the three experimental animals in which only the three left maxillary anterior teeth had been covered with the onlay, the periodontal membrane of the onlayed teeth differed markedly from the periodontal membrane of the maxillary teeth on the right side which were without the onlay.

In the onlayed teeth, the periodontal membrane was divisible into a comparatively narrow zone adjacent to the alveolar bone and a broad zone which occupied the remainder of the distance to the root surface. In some areas, the periodontal membrane communicated with broad marrow spaces of the alveolar bone (Figs. 3, 4 & 5). Adjacent to the bone, the periodontal membrane was devoid of principal fibres and was richly cellular, with polyhedral cells, numerous multinucleated osteoclasts and dilated blood vessels (Fig. 5). The broader zone of the periodontal membrane consisted of densely packed fibre bundles with closely arranged fibroblasts. The fibre bundles were separated by dilated blood vessels.

Along its entire surface in relation to the periodontal membrane, the alveolar bone was irregularly eroded and presented contiguous cove-like indentations with partially enclosed multinucleated osteoclasts (Fig. 5). In some sections, irregularly shaped spine-like projections of partially resorbed bone extended into the periodontal membrane. There was evidence of bone formation in isolated areas of the surface of the alveolus, along the endosteal surface of contiguous marrow spaces and along isolated trabeculae of bone on surfaces opposite to those undergoing resorption.

The cementum of the onlayed teeth was slightly narrower than that of the adjacent teeth without onlays or the teeth of the control animals. This difference was particularly evident in the linguo-apical area of the root. The thin layer of cementoid and adjacent cementoblasts which covered the entire root surface in the control animals was absent in the linguo-apical area of the onlayed teeth and there were oc-

Fig. 2. Radiograph showing onlay in position on the left maxillary anterior teeth. The numbered central and lateral incisors are those shown in Fig. 3.

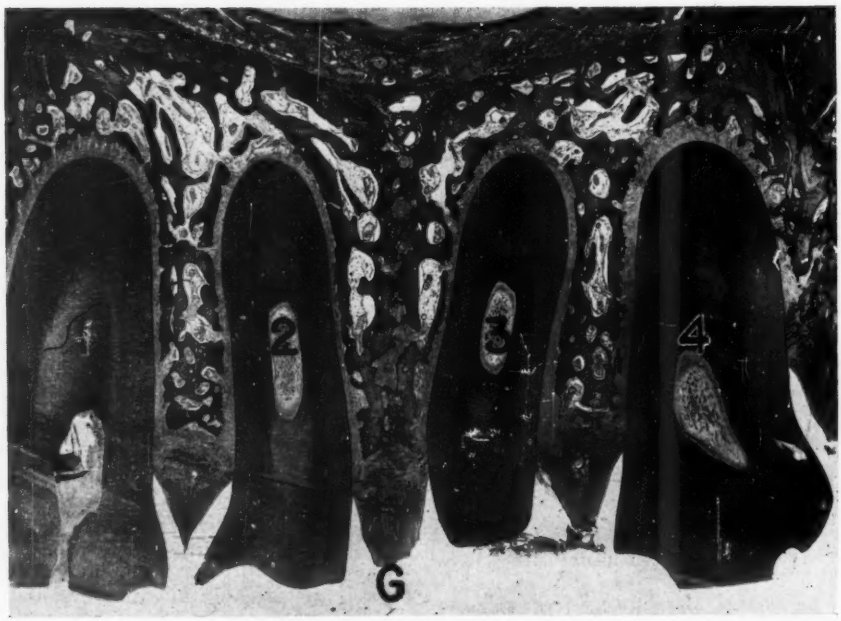

Fig. 3. Survey section through the right (1 and 2) and left (3 and 4) maxillary central and lateral incisors of dog at the end of 34 day experimental period. Teeth 3 and 4 were covered by the onlay. The enamel was removed in the preparation of the section. Note that the cemento-enamel junctions of the non-onlayed teeth are at a level which is incisal to the cemento-enamel junctions of the onlayed teeth. Hematoxylin and Eosin. Orig. Mag. X 12.

casional semi-lunar areas of cemental resorption which extended to involve the dentin.

The periodontal membrane of the anterior teeth without onlays was densely fibrillar with numerous fibroblasts and a moderate distribution of dilated blood vessels (Fig. 6). Bordering the alveolar bone, the fibrils were arranged as densely packed projections perpendicular to the bony surface and extending approximately one half the distance to the tooth (Fig. 7). In this zone, the linear outlines of the individual fibrils were not clearly discernible. Instead, the fibrils were clumped in wide bands which approached the texture of homogeneous matrix. The fibroblasts appeared to be enclosed within clear cut lacunar spaces in the matrix. The contiguous bone surface was lined with prominent osteoblasts in a linear arrangement and partially enclosed

Fig. 4. View of the root of the left maxillary lateral incisor and adjacent tissues from serial section of area shown in Fig. 3. Note the prominent blood vessels and direct communication of the periodontal membrane with the enlarged marrow spaces of the alveolar bone. Hematoxylin and Eosin. Orig. Mag. X 48.

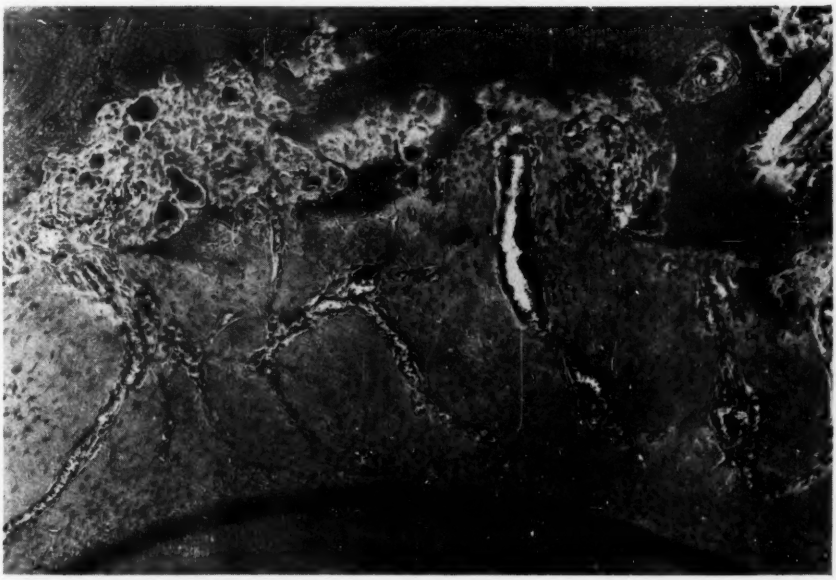

Fig. 5. Detailed study of the apical area of the left maxillary central incisor from serial section of area shown in Fig. 3. The zone of the periodontal membrane is richly cellular and contains numerous multinucleated osteoclasts adjacent to the eroded bone margin. The remainder of the periodontal membrane consists of densely packed fiber bundles and intervening blood vessels.

in osteoid (Fig. 7). In the zone of the periodontal membrane in relation to the teeth, the fibrils were heterogeneously arranged and blended imperceptibly with a prominent layer of cementoid on the root (Fig. 7).

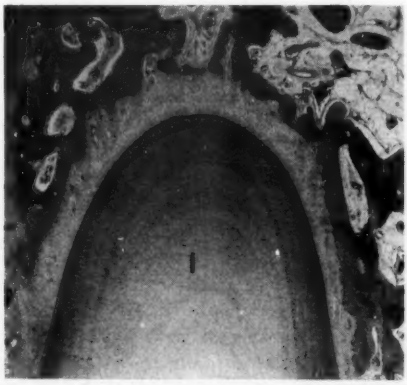

Fig. 6. View of the root of the right maxillary lateral incisor and adjacent tissues from serial section of area shown in Fig. 3. Note the uniformly fibrillar structure of the periodontal membrane. Hematoxylin and Eosin. Orig. Mag. X 48.

Gingiva. In both the control and experimental animals the gingiva presented chronic inflammation. The connective tissue of the gingiva was hyperemic, edematous and densely infiltrated with plasma cells and lymphocytes. The epithelium was slightly thickened, with acanthosis and proliferation of rete pegs into the underlying connective tissue. There was no appreciable pocket formation or proliferation of the epithelium in the area of attachment in either the control or experimental animals.

In the experimental animals, comparison of the gingiva in relation to the onlayed teeth and the adjacent teeth without onlays was particularly noteworthy. The teeth without the onlays extended further incisally than those upon which onlays had

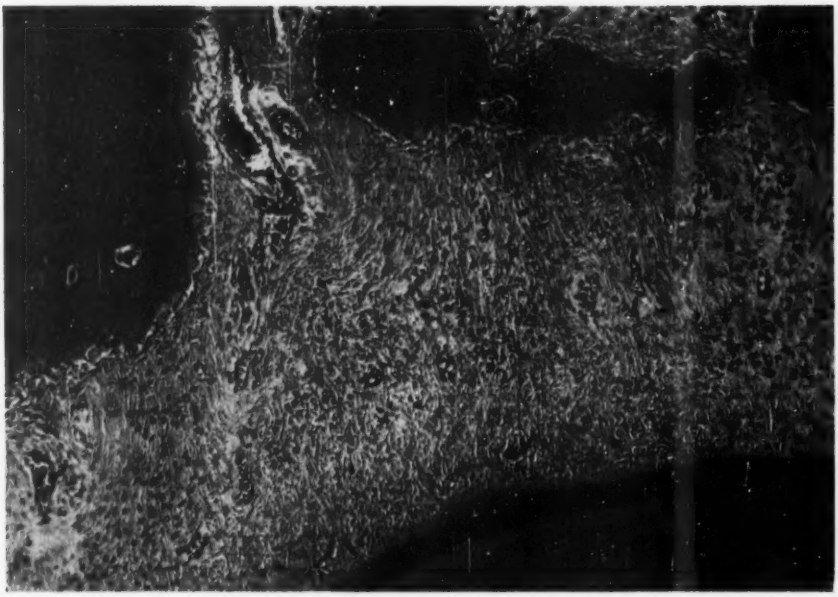

Fig. 7. Detailed study of the apical area shown in Fig. 6. Note the perpendicular arrangement of the fibrils in the periodontal membrane extending from the surface of the bone. The fibroblasts in this zone appear to be enclosed in lacunar spaces. The margin of the bone is lined with osteoblasts. Note the heterogeneous arrangement of the fibers in relation to the cementoid on the root surface. Hematoxylin and Eosin. Orig. Mag. X 128.

been placed. As a result, there was a difference in the level of the cemento-enamel junctions in the respective groups (Fig. 3). This difference was clearly visible in mesiodistal sections through the interdental space between the right (without onlay) and the left (onlayed) maxillary central incisors (Fig. 8). The epithelial attachment was at the cemento-enamel junction on both the onlayed teeth and teeth without onlays (Fig. 8). The course of the subjacent gingival fibers was altered in compliance with the difference in the levels of the onlayed and non-onlayed teeth. The transseptal fibers extended obliquely between the teeth rather than in the usual horizontal direction (Fig. 8).

DISCUSSION

It was the purpose of this study to investigate the specific question as to whether trauma from occlusion initiates periodontal pocket formation. This aspect of the microscopic findings will therefore be discussed in detail. The effects of trauma from occlusion upon the periodontal membrane, alveolar bone and cementum which have been dealt with extensively in the literature,[1] and noted in the introductory remarks, will be referred to only in so far as they may be pertinent to the problem of the initiation of pocket formation.

Under the conditions of this experiment, trauma from occlusion did not initiate the formation of periodontal pockets. Forces of sufficient severity to produce notable changes in the periodontal membrane and extensive resorption of alveolar bone, produced no deepening of the gingival sulcus or change in the position of the epithelial attachment on the tooth surface. Nor was there any evidence of proliferative change in the epithelial attachment, or detachment of epithelium from the tooth surface.

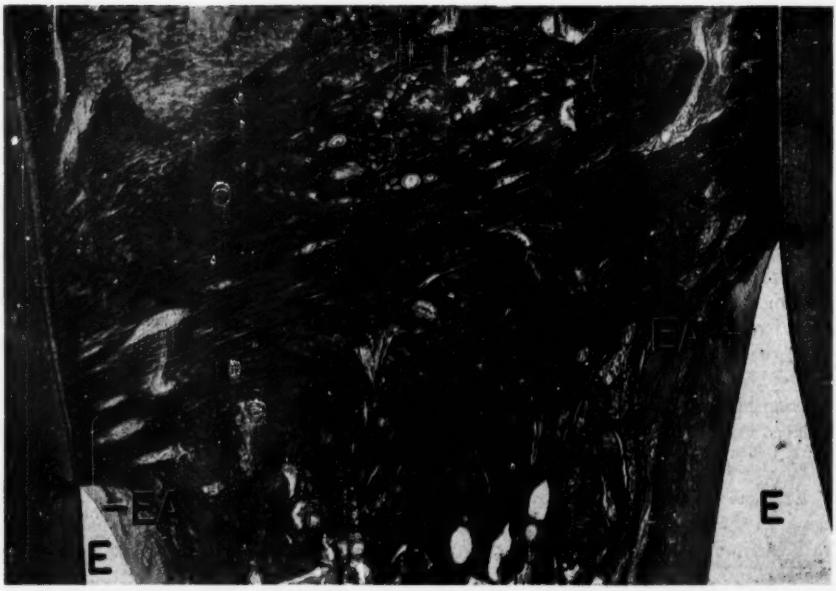

Fig. 8. Detailed study from serial section of area G in Fig. 3 showing the interdental space between right and left maxillary central incisors. The enamel (E) was removed in preparing the section. The difference in the levels of the cemento-enamel junctions is marked by the arrows. Note that the base of the epithelial attachment (EA) is in apposition with the subjacent gingival fibers and is located at the cemento-enamel junction on both teeth. The oblique course of the transseptal fibers is clearly shown. Mallory's Aniline Blue Stain. Orig. Mag. X 80.

It is noteworthy that in both the control and experimental animals the gingiva presented extensive chronic inflammatory involvement. The fact that periodontal pockets did occur when trauma from occlusion was introduced under such circumstances emphasizes the negative role of trauma in the initiation of periodontal pockets.

The observation that trauma from occlusion does not initiate periodontal pocket formation should not be enlarged into the broader generalization that trauma is in no way a factor in the overall process of pocket formation. It should be borne in mind that our experiment was concerned only with the initiation of periodontal pockets. The equally important consideration of the manner in which trauma from occlusion may contribute to the deepening of a periodontal pocket once it has been started was not investigated. From the presented findings it would therefore be presumptive to hypothesize regarding the role of trauma in any phase of the overall process of pocket formation other than that of the initial stage. Nevertheless, the findings leave us no alternative but to question the validity of the popular assumption that trauma from occlusion, in itself, is an etiologic factor in the initiation of periodontal pocket formation.

In seeking for a possible explanation of the findings, attention is attracted to the relationship of the epithelial attachment to the underlying gingival fibers and the location of these structures on the tooth surface. In the control animals, the base of the epithelial attachment terminated close to the cemento-enamel junction and the gingival fibers were embedded in the cementum immediately subjacent to the

epithelial attachment. In the experimental animals, in which only three of the maxillary anterior teeth were covered with the onlay, the relative positions of the onlayed teeth to the anterior teeth without onlays was altered at the end of the thirty four day experimental period. The teeth without onlays extended further incisally than the onlayed teeth. The extent to which this change in position of the teeth resulted from depression of the onlayed teeth or incisal movement of the teeth without onlays would require cephalographic study. However, the microscopic findings in regard to the epithelial attachment—gingival fiber relationship in the altered positions of the onlayed and non-onlayed teeth were definitive.

Examination of the interdental gingiva between the onlayed and non-onlayed central incisors indicated that the epithelial attachment and gingival fibers retained their positions in the region of the cementoenamel junction despite the change in the relation of the teeth to each other. The base of the epithelial attachment was at the cervical margin of the enamel and in apposition with the gingival fibers. The transseptal fibers deviated from their usual horizontal course and extended obliquely so as to accommodate the altered levels of the teeth and still retain their normal relationship to the base of the epithelial attachment.

This tendency to retain or restore normal anatomic relationships in the presence of an alteration in the environment was manifested in the underlying tissues as well as in the gingiva. In the teeth without onlays, which appeared to have moved incisally, there was a widening of the periodontal membrane with a subsequent attempt to restore the normal anatomic relationship of the periodontal membrane, alveolar bone and tooth by apposition of alveolar bone and cementum. In relation to the onlayed teeth, there was accentuated resorption of alveolar bone which permitted the teeth to move into a position in which the occlusal forces would exert a salutory effect upon the periodontium. There was also apposition of bone to strengthen trabeculae of the alveolus which had been weakened by resorption.

Subject to the qualifying limitations imposed by all speculative hypotheses, some explanation for the presented findings in respect to trauma from occlusion and the gingiva may lie in the above analysis. It suggests that the response of the gingival tissues to increased functional force is not in the direction of pathologic change but that it is determined by an inherent tendency to retain or restore the normal anatomic relationships of the gingiva to the tooth. The finding that trauma from occlusion did not initiate pathologic pocket formation, but instead was accompanied by the persistence of an epithelial attachment which was normal in its relationship to the tooth and subjacent tissues could be considered as an expression of this inherent tendency.

SUMMARY

Under the experimental conditions induced by cementing onlays on the anterior teeth of dogs, trauma from occlusion did not initiate the formation of periodontal pockets.

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An Overall Philosophy of Treatment of Periodontal Disease and Case Analysis

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THE overall philosophy of any dental practitioner in the treatment of periodontal disease is embodied in the views he promulgates as a result of his training, skill, personal insights, knowledge and experience in the treatment of periodontal ills. The objective of this paper is not to assay controversial forms of treatment or to delve deeply into any single aspect of the science of periodontology but, rather to reveal a philosophy by showing how a case may be analyzed in the preparation for and during the progress of periodontal treatment. It is felt that this approach might prove to be of more value than a discussion of a particular phase of periodontal diagnosis or treatment, which would of necessity presume that the overall philosophy of diagnosis and treatment of periodontal disease is a well-worn and most useful tool in the chest of knowledge of every periodontist. A re-examination and re-evaluation of procedures are never entirely without merit.

The importance of a thorough understanding of the basic sciences cannot be overstressed. The many and varied pathological changes seen in the oral cavity can never be diagnosed with accuracy unless the normal anatomy, histology, and physiology of the parts are well known. Many of the changes seen in the mouth cannot be correctly interpreted or related, except in the light of our knowledge of the basic biochemical and bacteriological actions and reactions. Every step of case analysis requires thoughtful consideration and intelligent decisions. This will not be possible unless the practitioner thoroughly understands the fundamental processes of the body in health and in sickness. Our very first assumption, therefore, is that the periodontist must at all times seize every opportunity to enrich and extend his knowledge of the sciences as they pertain to periodontal disease.

Exactly what is case analysis? It is the art of considering thoroughly all of the factors which have produced the existing condition or conditions in a patient's mouth, plus the institution of a flexible but definite plan by which the mouth may best be restored to health. Gingivitis and periodontitis comprise approximately 90 per cent of all of the periodontal diseases.[4] The case analysis and treatment plan discussed hereafter is particularly applicable to these conditions.[1] In a more general sense, nevertheless, periodontosis, atrophy, and other disturbances may be included also.

The first step in case analysis and treatment is examination, recording and treatment planning. All information relating to conditions of the teeth, the gingivae, and soft tissues of the mouth, and all data relating to the personal and family history should be carefully recorded. Special attention should be focused upon any systemic condition which may be present such as: subclinical avitaminosis, diabetes, blood dyscrasias, syphilis, faulty mineral metabolism, and any other general, dibilitating disease.

It is important to remember that no matter how completely all local irritants are removed, how skillfully and thoroughly all pockets are eliminated, or however effective has been the instruction in the correct institution of home care during the treatment process, only temporary or partial success can be attained if the general health of the patient is poor. Too many practitioners have become frustrated and discouraged by failures with cases where the principal causitive factors were systemic

when they had otherwise done a fairly good job in the correction of the local conditions involved. They neglected to discover and correct all systemic conditions which modify or prevent adequate response of the tissues to local treatment. Many hours of fruitless labor might have been averted if the examination had been comprehensive or thorough enough to reveal the systemic condition responsible for the poor health of the tissues. An important part of our concept of the overall management of periodontal patients is based, therefore, upon an evaluation of their general health.

While the depth of pockets is being recorded and the mobility of the teeth is being measured with the purpose of determining the prognosis of individual teeth, pitfalls await the one who is bound by inflexible rules. For example, a tooth with two or more millimeters of movement ordinarily is considered hopeless, as is the case also of a tooth which can be depressed within its socket. But, mitigating factors, such as the presence of acute lateral abscesses, or the manner in which the remaining bone is distributed in each tooth examined, must be considered with careful discrimination.[2] It is well to remember that neither the depth nor the configuration of a periodontal pocket can be determined from the x-ray, alone. Periapical films are especially unreliable. Bitewing x-ray films are better, but the bottom of the pocket never coincides with the bony alveolar margins. There is always soft tissue between the pocket and bone.

Usually, it is believed that a *minimum* of one-half of the total periodontal membrane attachment must remain after treatment in order to attain satisfactory results. Too often the estimate of how much attachment is left is made from x-rays alone, but the attached soft tissues containing bone matrix cannot be seen in x-rays. Too often an estimate of the attachment is made considering the entire length of the tooth or the entire length of the root. Neither is correct.

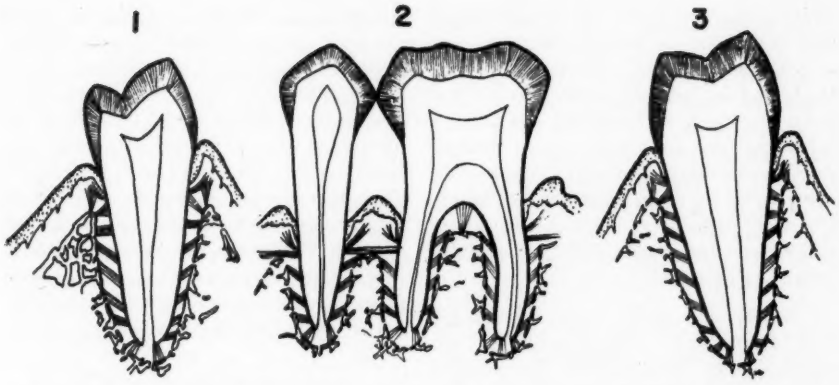
Many teeth have been lost as a result of snap judgment from viewing an x-ray, or because of a misunderstanding of how much support the tooth had originally as compared with the existing condition. Furthermore, the estimate of how much attachment is left is often made without due consideration to the lingual and buccal or labial bone present. Rarely is the loss of bone in these areas as great as it is in the interproximal areas. (See Figures 1, 2 and 3.)

It is necessary constantly to think in three dimensions when making deductions from x-rays. Actually the continuous deposition of cementum upon the root surfaces of the teeth increases the diameter and length of their roots. Therefore, the total surface area in which fibers may be attached is greater with increasing age. This is nature's way of compensating for continuous eruption and occlusal wear.

As soon as the examination, recording and treatment planning are completed, we proceed then with the second step, namely, the removal of all hopeless teeth. Judgment on questionable teeth is deferred until later in the treatment. This is especially important in cases where the teeth in question have been exposed to severe occlusal trauma. Such teeth should be taken completely out of occlusion at this time to give the tissues involved a chance to recover. However, all teeth that are clearly beyond the stage of successful treatment should be removed without delay.

The next step in our procedure consists of excavating all deep carious cavities and protecting them with medicated, temporary fillings. It would indeed be odd therapy to manage somehow to save the supporting structures of a tooth only to lose additional tooth substance or the tooth itself from advancing caries. Proper treatment of the hard and soft structures of the teeth, therefore, becomes an important role in the total picture of periodontal therapy.

We are now prepared to proceed to the "so-called" routine treatment, which consists of (1) thoroughly scaling and polish-



Figures 1 and 3 diagrammatically show bucco-lingual sections of a second bicuspid and first molar of the same arch with their high buccal and lingual attachments, while Figure 2 shows the condition of the same teeth in mesio-distal section.

ing each surface of the crowns and roots of the teeth involved, (2) stimulating the circulation in the gingival tissues, and (3) instructing the patient in the home care of the teeth and gums. The best that any dentist can possibly expect of competent treatment will be to get the tissues in a healthy and strong state. The patient must keep them that way by constant, diligent and intelligent home care.

Hirschfeld,[3] Orban,[5] and others have estimated that about 75 per cent of the success achieved in periodontal therapy depends upon the patient's home care. Yet how much time does the average dentist devote to the teaching of toothbrushing, gingival massage, and interproximal care? Far too little! A patient cannot be taught adequate home care by the over-simplified method of lectures, films, or demonstrations on models alone. These devices are important but demonstration in the patient's mouth is absolutely necessary. The dentist should demonstrate *in the patient's mouth* how the teeth and gums should be brushed, and the reason for each procedure, while the patient watches in a large hand mirror. The patient then brushes while the dentist watches and corrects mistakes. The patient is requested to bring his brush and rehearse the procedure at each visit. Corrections or additions to the technique are made by the dentist-teacher during each lesson until the method has come to be a well-established habit in the hands of the patient. Much of therapeutic value of a periodontist's service will depend upon his skill, patience, and time devoted to patient instruction.

Many fail to realize that it is normal (at least in so far as incidence is concerned) for the interproximal areas to become food traps in adults as the papillae recede with age. Periodontal diseases make these food traps larger. We contend that no method of brushing adequately cleanses the interproximal areas once the gingival papillae have receded. Hence, the use of adjuncts such as rubber tips, dental floss, toothpicks, stimudents, or pipe cleaners must be taught to the patient in much the same manner as toothbrushing was described. Painstaking effort in this direction will reap rich dividends in the form of tightening of the teeth, reduction of pockets, bone formation, and little or no recurrence of the disease.

The next step in treatment is the removal of all remaining mechanical irritants such as overhanging fillings, poor bridges, shell crowns, and misfitting restorations of any form. Let it not be forgotten that every time a filling, crown, bridge or denture is placed, there is being practiced either good or bad periodontal treatment. Permanent

replacements for the restorations removed may be deferred until the supporting tissues are again healthy. However, the restorations are placed immediately if the operator believes that such replacements will aid recovery of the supporting tissues. Bridges or removable partial dentures are usually contraindicated in the presence of questionable teeth remaining in the mouth. These conditions require individual and carefully arrived at judgment.

The treatment of any dietary deficiencies, first by artificial supplementation and eventually by correction of the natural dietary intake is now decided and carried out. If there had been clear-cut evidence during the examination of a gross dietary deficiency, this would have been corrected as the very first step in treatment. More often, however, there is a sub-clinical lack for which specific therapy aimed at correction can profitably be instituted at this time. Sometimes, however, the expected improvement in the tone and quality of the periodontal tissues does not occur. If we are certain of the thoroughness of removal of all local irritants, and if we are confident of the adequacy of the patient's home care, then the systemic examination should be painstakingly repeated. All consultations with the patient's private physician, nutritionist, or other specialists deemed necessary in checking for systemic disorders should be arranged. While it is no doubt true that local conditions act as the exciting cause of 90 per cent of the periodontal diseases, the general systemic condition of the patient plays a major role in the success of any treatment.

Attention is directed next to the correction of occlusal disharmony and the establishment of a gliding occlusion. Centric relation, protrusive excursion, protrusive position, and the lateral excursions should be brought into harmonious relationships. The major objective in equilibrating the occlusion should be to divide the stresses of occlusion among the remaining teeth in direct proportion to their capacity to bear the forces of mastication. In other words, stronger teeth should accept the "brunt of the load" of mastication while teeth that have lost considerable attachment should not be overtaxed. Many cases, that have been successfully managed otherwise, fail ultimately because of insufficient attention to this simple, physical principle in treatment. It is advisable to observe and re-check an occlusion that has been equilibrated until a reasonably static relationship is established. Sometimes the shifting of the teeth following grinding presents new problems for correction within several days or weeks, later.

At this point in the treatment, all questionable teeth that have not responded favorably should now be removed. If at any time during the course of treatment a questionable tooth becomes hopeless, it should be removed without hesitation. In this way, unnecessary waiting for healing of the wound before replacement is avoided and the whole matter of complete mouth rehabilitation is enhanced.

Pockets that remain at this stage will usually have to be removed by surgery or chemical cauterization. It is not so important what method is employed for the removal of pockets as it is that they be carefully eradicated, whereby a hygienic condition is made possible. Whichever method of pocket removal is attempted, the success will depend largely upon the post-operative care by the dentist and patient while the tissues are healing at a new level. Frequently this step will have been performed immediately following the removal of local irritants. The determining factor here will be the judgment of the clinician as to whether local treatment alone might result in ample reduction or repair of the pocket areas.

It is essential that temporary and permanent stabilization of weakened teeth be effected wherever indicated. This procedure includes the replacement of missing teeth. Proper sharing of the occlusal load may often be attained by the judicious use of interlocking inlays, interlocking bridges,

and circumferential cast splints. When partial dentures are indicated, as many teeth as possible should be used in sustaining the forces brought to bear upon the appliances. Where the case was referred by another dentist, the patient is usually referred to him for this work with a careful prescription and explanation from the periodontist. (A consultation for this is best procedure.) The patient should be seen when this work is completed for the purpose of re-checking the occlusion and also the restorations which were placed.

A program for regular prophylaxes and periodic check-ups should be discussed and arranged before the patient is dismissed. He should certainly be apprised of the fact that his investment cannot be protected properly without regular dental care to prevent recurrence of the original condition. Furthermore, it is important that the patient be made to realize that the overall objective of treatment is not merely to arrest the disease process and restore the mouth mechanism to normal function, but to establish a general physical condition which will lead to a vibrantly healthier, happier life.

SUMMARY

An attempt has been made here to reveal our overall philosophy in the treatment of periodontal disease through a statement of case analysis and treatment procedure. This procedure, too, has been set forth in steps in the order of usual importance. Too much stress, however, cannot be placed upon the need for constant flexibility in moving from step to step. The conditions of each case will determine whether occlusal equilibration is needed first, or whether immediate medical consultation is more urgent. It must be kept in mind that treatment of no single factor can cure periodontal ills; rather, it is the correction of all of the inter-related conditions which produced the disease, combined with a program of adequate home care to prevent its recurrence. This alone will result in successful periodontal therapy.

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Microscopic Demonstration of Tissue Reaction Incident to Removal of Subgingival Calculus

by JENS WAERHAUG,* PH.D., D.D.S., *Minneapolis, Minnesota*

IN a previous work,[1] mainly based on surface investigation of stained extracted teeth, it has been shown that the epithelium can re-join the tooth surface in areas earlier covered by subgingival calculus in the same way as a normal epithelial cuff (epithelial attachment) joins the tooth surface. However, good histological pictures were not provided due to the difficulty of obtaining favorable biopsy specimens from human beings. It has further been demonstrated[2] that pockets without subgingival calculus usually are sterile, while pockets with subgingival calculus always are infected with bacteria. Finally it has been shown[2] that bacteria inserted into healthy pockets will disappear within 48 hours, and that pockets with subgingival calculus can be sterile again if the calculus is thoroughly removed.

The object of this experiment was to provide good histological pictures showing the healing after removal of subgingival calculus.

MATERIAL AND METHOD

Human material is not well suited for an experiment of this kind because it is impossible to remove a biopsy specimen without moving the gum tissue rather extensively in relation to the tooth surface, which results in the pattern of the soft tissue being altered. It is also difficult to avoid extensive artificial splits between the tooth and the soft tissue, and these make the interpretation of the sections uncertain. It was, therefore, decided to use animals where these complications do not exist. Supragingival calculus is a common finding in old dogs and monkeys. Subgingival calculus is, however, seldom found as the gingival margin usually recedes with the same speed as the calculus grows apically. This is in contrast to findings in human beings. Finally an old dog was found with calculus about 1.5-2.0 mm below the gingival margin on the buccal surfaces of the four cuspids. Two of these teeth were thoroughly scaled and the surfaces polished with pumice. On the two others the calculus was not touched so that these pockets might be used as controls. The dog was sacrificed after 90 days. The jaws were taken out with great care and fixed in 10 per cent formalin. The specimens were embedded in celloidin and stained with hematoxylin-eosin.

OBSERVATIONS

Clinical observations before the scaling was done revealed some bleeding from the pockets when pressure was applied at the gingival margin. The calculus was firm, as in man, and covered on the surface by a soft slimy layer. After the removal of the calculus the bleeding subsided and normal conditions were observed after two weeks. At the end of the observation period a certain amount of plaque had been re-formed above the gingival margin on the scaled teeth.

On the control tooth where no scaling had been done, large amounts of subgingival calculus reach more than halfway down the pocket (Figure 1). Degenerated epithelial cells border directly upon the bacterial plaque on the surface of the calculus (see higher magnification to the left), and a great number of rete pegs have proliferated into the subjacent connective tissue which is densely infiltrated by lymphocytes and plasma cells. Epithelial cells have migrated

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Fig. 1. Dog 15. Untreated control tooth. Great amount of calculus (CA) reaching more than halfway down the pocket. Epithelium corresponding to calculus shows rete pegs, and underlying connective tissue is inflamed. In higher magnification to the left bacterial plaque on surface of calculus is seen to lie in direct contact with epithelium. Epithelium degenerated and connective tissue infiltrated by lymphocytes and plasma cells. In higher magnification to the right inflammatory foci (INF) are seen below cemento-enamel junction (CEJ). Bottom of clinical pocket (BP) deepened below cemento-enamel junction.

below the cemento-enamel junction (see higher magnification to the right), and inflammatory foci, INF, are seen near and below the bottom of the pocket.

Figure 2 shows the tooth from which subgingival calculus had been removed. The pocket depth, measured during the scaling, was found to be about three millimeters. The distance from the gingival margin to the cemento-enamel junction is 3.5 mm. There is very little subgingival calculus (CA). The epithelium which joins the enamel nearly to the gingival margin shows a few rete pegs corresponding to the calculus, and there is some round cell infiltration in the connective tissue immediately below (see higher magnification to the left). The inflammatory reaction decreases rapidly and is absent halfway down the pocket (see higher magnification to the right). The epithelial cuff (epithelial attachment) here is normal and joins tightly to the enamel surface.

DISCUSSION

There is a conspicuous difference between the tissue reaction in Figures 1 and 2. Thanks to very careful handling of the specimens during the preparation process the artificial splits commonly found between the calculus and the soft tissue or within the soft tissue have not developed in Figure 1. The bacterial plaque on the surface of the calculus here borders directly upon the pocket epithelium, and the bacteria and their toxins have caused a degeneration of the epithelium and a severe inflammation in the connective tissue. This inflammation is most pronounced in the area corresponding to the calculus, and decreases toward the cemento-enamel junction. But inflammatory foci can be traced even below the alveolar crest. There is every reason to believe that the conditions were about the same on the tooth shown in Figure 2 at the time the subgingival calculus was removed. The depth of this pocket, when measured at the time of operation, was about 3 mm. The distance from the gingival margin to the cemento-enamel junction is 3.5 mm. This confirms earlier observations that the bottom of the clinical pocket is to be found at the deepest point of the "epithelial attachment." The calculus reached in this case 1.5 to 2 mm below the gingival margin. This means that a new and normal epithelial cuff has

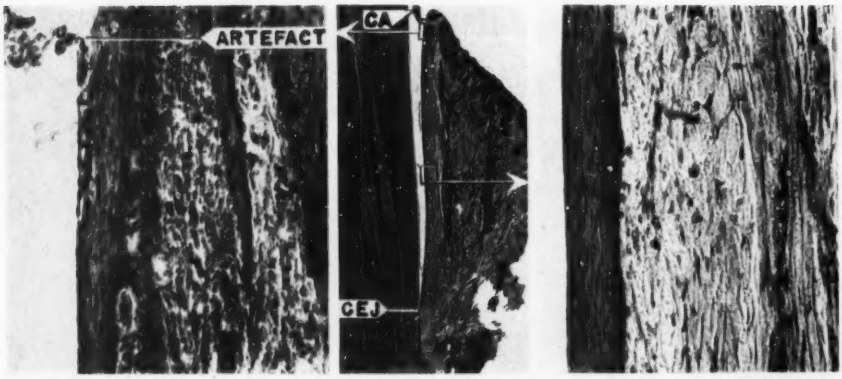

Fig. 2. The same animal as Fig. 1. Three months before death subgingival calculus reaching 1.5 to 2.0 mm below gingival margin removed. Depth of clinical pocket, measured during scaling, about 3 mm. Distance from gingival margin to cemento-enamel junction, 3.5 mm. Normal epithelial cuff covers enamel surface nearly to gingival margin. Small amount of calculus (CA) reaching 0.3 mm below gingival margin. Slight inflammation in connective tissue below gingival margin. Inflammation practically absent in center part of pocket and below it. No deepening of clinical pocket below cemento-enamel junction (CEJ).

been formed in the area previously covered by subgingival calculus. It is remarkable that most of the rete pegs in the epithelium and most of the inflammation in the connective tissue have disappeared. The slight round cell infiltration in the gingival margin is caused by the small amount of subgingival plaque which has lain in contact with the soft tissue. This inflammation decreases rapidly within the first millimeter below the gingival margin and is practically absent in the center part. On the control tooth (Fig. 1) inflammatory foci are seen far beyond the cemento-enamel junction, and these foci have broken down the periodontal fibers just below the epithelial cuff allowing the epithelial cells to proliferate along the root. The removal of subgingival calculus has, consequently, served two favorable purposes: (1) The width of the soft tissue cuff lying in direct contact with the bacterial plaque on the surface of the calculus has been reduced, which has resulted in a decrease of the inflammation. (2) The point of attack of the bacteria has been moved to a greater distance from the cemento-enamel junction, with the result that the far effect of the existing reduced inflammation does not reach beyond it, thus causing a deepening of the pocket.

The prerequisite for a readaptation of healthy epithelial cells to the tooth surface in areas earlier covered by subgingival calculus is that no bacteria be retained on the tooth. Earlier experimental investigations[2] have shown that concentrated cultures of virulent bacteria inserted into healthy pockets will not remain there for any length of time, as they are squeezed out again or taken care of by the polymorphonuclear leukocytes. The same renovation process apparently takes place after removal of subgingival calculus if the removal is complete. Thus, bacteria do not seem to be able to remain below the gingival margin if they are not retained in the filamentous stroma of Actinomyces and Leptothrix, which grow in firm colonies. Such bacterial plaque has been formed above the gingival margin in Figure 2, and it has also grown a very short distance below it. There is reason to believe, however, that the formation of this plaque could have been prevented if the tooth had been brushed thoroughly every day.

This experiment confirms earlier observations on human material that a complete healing of the soft tissue with the establishment of a sterile pocket may be the result of a thorough removal of subgingival cal-

culus. It also shows the importance of this treatment.

CONCLUSIONS

A complete removal of subgingival calculus will, under favorable conditions, lead to a re-formation of a normal epithelial cuff in areas earlier covered by the calculus, and it may result in a more or less complete disappearance of the inflammation caused by the calculus.

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Observations on the Composition and Metabolism of Normal and Inflamed Gingivae*

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EARLY investigators[1,37] reported that changes in the glycogen content of gingival tissue occur after the onset of inflammation and suggested that a greater glycogen content might increase the resistance to gingival disease. Other workers have claimed that well keratinized gingival epithelium gives protection against gingival disease.[8] Still others have linked glycogen content and keratin production in the process of wound healing.[6] These observations suggested a possible mechanism by which the gingival glycogen might give protection.

Accordingly this study was undertaken in an attempt to elucidate changes in metabolism which occur when gingival tissue becomes inflamed. Histochemical methods were utilized for the demonstration of glycogen and other carbohydrates, of keratin, alkaline phosphatase and nucleic acid.

*Portion of this work was done with the aid of a grant from the National Health and Medical Research Council of Australia.

**Dental Board of N.S.W. Research Officer. From the Institute of Dental Research, United Dental Hospital of Sydney, Australia.

MATERIALS AND METHODS

MATERIALS

Gingivae which appeared normal on close clinical examination were studied from eight patients; these were mainly children from whom teeth were extracted in the course of orthodontic treatment. Gingivae also were studied from seven patients suffering from very mild marginal gingivitis; likewise biopsy material from forty-one patients who suffered from gingivitis or periodontitis. In some of these forty-one patients there were obvious systemic disturbances, such as occurred in pregnancy, puberty and diabetes, which might act as modifying factors, and in all cases of gingivitis and periodontitis sources of local irritation, such as trauma or calculus or poor oral hygiene, were present.

METHODS

Tissue from both the gingival margin and the interdental papilla was removed at biopsy under novocaine anesthesia. The tissue was dissected and fixed im-

mediately in Carnoy fluid (30 minutes), ice-cold acetone (24 hours) or a 4 per cent lead acetate-10 per cent formalin solution (24 hours). Paraffin-embedded sections, 7μ thick, were prepared.

STAINING TECHNIQUES

Harris' haematoxylin-eosin and Masson's trichrome methods were used to show histological detail and Gram's stain (Lillie modification) for the demonstration of microorganisms in the tissue.

Keratin. For the demonstration of sulphydryl groups and keratin the most satisfactory procedure was the Lillie modification[22] of the Chèvremont and Fréderic prussian blue stain (20 minutes). Control sections were exposed for 48 hours to saturated phenyl mercuric chloride in 80 per cent ethyl alcohol to block the sulphydryl groups. Disulphide groups were detected by pretreatment of the sections for 10 minutes with 5 per cent potassium cyanide. Carnoy fluid and ice-cold acetone were satisfactory fixatives for tissue to be treated by this prussian blue technique.

Carbohydrate. The presence of glycogen, mucopolysaccharides and glycoproteins was demonstrated by the Hotchkiss technique,[19] using Carnoy-fixed material. The technique of Hotchkiss (aqueous solutions) and Staple's suggested modification of this technique (alcoholic solutions)[33] were both used. Control sections were incubated at 37° C. for 1 hour in the presence of amylase (saliva) before staining, in order to distinguish glycogen from other reacting substances.

The Hotchkiss and the prussian blue techniques were combined to reveal the location of glycogen deposits in relation to keratin, the sections being stained first for keratin and then by the Hotchkiss technique (aqueous solutions). When the staining procedures were carried out in this sequence, specificity of the stains was apparently not affected.

The acid mucopolysaccharide granules in mast cells were stained by a toluidine blue staining technique recommended by Kimball.[21] Basic lead acetate-formalin fixation was found the most satisfactory. The mast cells present in the connective tissue of the whole section were counted and expressed as number per unit area.

Alkaline Phosphatase. Alkaline phosphatase was demonstrated by the Gomori[16] technique applied to tissue fixed in ice-cold acetone. Paraffin-embedded sections were incubated in sodium glycero-phosphate for 45 minutes.

Nucleic Acids. To distinguish the nucleic acids, sections were stained with 1 per cent toluidine blue at pH 5.0 for 3 minutes and quickly washed in 95 per cent alcohol. In gingival tissue acid mucopolysaccharides also possess basophilic properties, so that specific extraction procedures were necessary in order to demonstrate the nucleic acids. Control sections were incubated for 12 minutes at 60° C. in 2N HCl to extract ribonucleic acid. Using Carnoy-fixed tissue it was found that this treatment removed the ribonucleic acid (R.N.A.) but did not remove detectable amounts of desoxyribonucleic acid (D.N.A.) or acid mucopolysaccharides. D.N.A. was stained by the Feulgen technique.[28] Control slides were stained to detect free aldehydes.

RESULTS

The histological and histochemical pictures of the inflamed gingivae were found to be very similar, whether the history revealed local disturbances as the only likely cause or systemic disturbances in addition to local. The interdental papillae showed changes similar to those in the gingival margins, although in a small number of specimens the papillae showed greater inflammatory reaction than the margins.

GENERAL HISTOLOGICAL DESCRIPTION

General histological description of the clinically normal gingivae showed five of the eight specimens were inflamed. Small

Fig. 1. Section from inflamed gingiva, stained to demonstrate glycogen. Deposits occur in the marginal epithelium but the crevicular epithelium is free of deposits (Hotchkiss stain: magnification approx. 35 X).

numbers of inflammatory cells were present in the connective tissue subjacent to the crevicular epithelium. The degree of inflammation was thus very slight and could be classified as "subclinical." In four of the seven specimens of gingiva classified clinically as showing *very mild* marginal gingivitis, diffuse and focal accumulations of chronic inflammatory cells were present. The remaining three specimens showed evidence of slight chronic inflammation.

In the clinically inflamed gingivae, subacute to chronic inflammation with infiltration by inflammatory cells, mainly lymphocytes and plasma cells, was observed in the subepithelial connective tissue; many mast cells were also present. Infiltration of the epithelium of the gingival crevice by leucocytes was always in evidence, together with slight destruction of tissue. Moderate hypertrophy of the interpapillary processes of the crevicular epithelium occurred. Vessels were dilated and there was some oedema together with slight disorganization of connective tissue.

GENERAL HISTOCHEMICAL PICTURE

Keratin. For this work keratinization is defined as the production of keratin identifiable histochemically and not merely hornification due to cytomorphosis. Tissue was considered keratinized if it showed the appropriate reaction to histochemical techniques, in addition to conforming to the histological criteria of Castenfelt:[8] absence of nuclear staining; flattened nuclei (if present) and flattened cells with acidophilic cytoplasm.

The superficial layers of the marginal epithelium were keratinized in all clinically normal and *very slightly* inflamed tissue, whereas eight of the forty-one specimens of inflamed gingiva were non-keratinized. Keratinization was never observed in the crevicular epithelium, normal or inflamed.

Carbohydrates. Glycogen: Clinically normal gingivae, free from invading leukocytes, were free from glycogen. Those showing subclinical inflammation contained a few granules of glycogen in the epithelium. In the inflamed gingivae, the epithelium contained much glycogen. Thus the amount seemed to be dependent on the degree of inflammation. The pattern of deposition of glycogen is constant: triangular areas in the superficial Malphigian layers and stratum

Fig. 2. Higher magnification of section shown in Figure 1. Deposition of glycogen in stratum corneum and the higher Malphigian layers of epithelium (Hotchkiss stain: magnification approx. 100 X).

corneum of the marginal epithelium; the apices of the triangles are in the deeper cells of the Malphigian layer, pointing towards the zone of connective tissue infiltrated with inflammatory cells; the bases of the triangles lie along the superficial layers of the epithelium (Figures 1 and 2).

No deposition of glycogen occurred in the crevicular epithelium or in the lymphocytes or plasma cells. A few granules of glycogen were present in the connective tissue of the inflamed gingivae.

Glycoprotein: In the superficial layers of the marginal epithelium of both normal and inflamed gingivae the glycoprotein of the intercellular substance stained heavily irrespective of the degree of keratinization. In the deeper Malphigian layers and in the basal layers the staining was faint, even very faint or absent in the basal layers. In the crevicular epithelium the staining was likewise very faint or absent.

The basement membrane of glycoprotein was mainly intact but in small isolated areas in thirty-seven of the forty-one specimens of inflamed gingivae it had apparently been dissolved.

The glycoprotein of the ground substance of the connective tissue was clearly seen, but in decreased concentration in the region invaded by inflammatory cells.

Acid mucopolysaccharide: Mast cells containing granules of sulphonated mucopolysaccharide were present in all specimens but there was a significant increase in numbers in the inflamed gingivae. The counts were: in clinically normal gingivae, 18.3 per sq. mm. (standard deviation = 14.3) and in inflamed gingivae, 47.0 per sq. mm. (standard deviation = 23.5). The difference is statistically significant.

Alkaline Phosphatase. In the normal gingiva alkaline phosphatase activity was

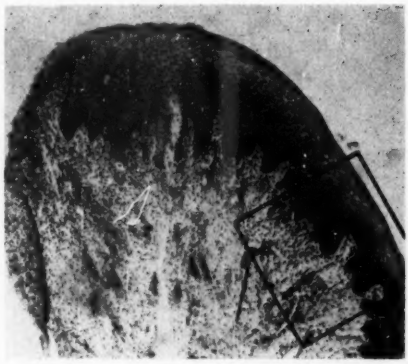

Fig. 3. Section from inflamed gingiva, stained to demonstrate nucleic acid content. Heavy staining in marginal epithelium but light staining in the crevicular epithelium (Toluidine blue: magnification approx. 35 X).

confined to the nuclei of the endothelial cells of the vessel walls, and in the inflamed gingiva to these nuclei and the inflammatory cells.

Nucleic Acids. The nucleic acid distribution was similar in normal and inflamed gingivae, except that, where infiltration of the epithelium by leucocytes occurred, D.N.A. was present in the

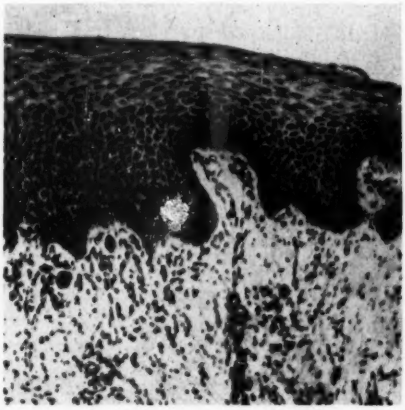

Fig. 4. Higher magnification of section shown in Figure 3. Heavy staining of nucleic acids in basal and lower Malphigian layers, inversely to deposits of glycogen shown in Figure 2 (Toluidine blue: magnification approx. 100 X).

nuclei of inflammatory cells and tissue cells. R.N.A. was present in large quantities in the cytoplasm of the basal cells of the gingival epithelium but became progressively less towards the superficial layers (Figures 3 and 4). The concentration of R.N.A. was always lower in all cells of the crevicular epithelium and in regions of the marginal epithelium which were infiltrated by leucocytes (Figures 5 and 6).

DISCUSSION

NORMAL GINGIVA

The criteria for "clinically normal" were very exacting and all gingivae classified as showing "very mild marginal gingivitis" would probably have been classified as normal by most clinicians. Histological examination later justified the use of such exacting criteria: only three of the eight specimens considered normal clinically were free of inflammatory cells and these were orientated in such a way that the crevicular epithelium and the subjacent zone which is most frequently invaded by leucocytes could not be examined. The other five specimens showed very slight infiltration of leucocytes. Some investigators[11] consider that in normal gingiva, as in the nasal mucosa and the tonsil, leucocytes are always present but Bernier[2] considers that their presence indicates "subclinical inflammation."

Keratin. The epithelium lining the gingival sulcus may *usually* be non-keratinized but no statement can be made as to whether or not the *normal* crevicular epithelium is keratinized until investigation of non-infiltrated crevicular epithelium has been carried out on human material removed along with the adjacent tooth.

The evaluating committee of the periodontal workshop[11] held in 1951 at the University of Michigan concluded that the epithelium lining the normal gingival crevice was not keratinized. This is surprising because no reference quoted by the committee supported the view that normal human crevicular epithelium was non-keratinized. Most of the histological observations related to the marginal epithelium, and those few that referred to the crevicular epithelium presented evidence of a keratinized epithelium.

Carbohydrates. Glycogen: No glycogen was observed in the normal gingivae. Wislocki and Sognnaes[36] and Engel et al.[12] noted small deposits of glycogen in normal gingiva but no details were given to show how carefully these cases were selected and, histologically normal gingival tissue being obtainable only rarely, it is possible that the tissue was slightly inflamed. It is possible also that in the freeze-dried specimens studied by these workers minute quantities of glycogen were observed which are not detectable in chemically fixed tissue.

Glycoprotein: The absence of stainable glycoprotein from the intercellular substance in the basal layers of the marginal epithelium and from the crevicular epithelium is of interest. This may indicate a different degree of polymerization of the intercellular substance in these regions. In the crevicular epithelium some destruction of the intercellular glycoprotein may have taken place where infiltration by leucocytes and other substances had occurred.

Acid mucopolysaccharide: The large number of mast cells present in gingivae is of interest in that these cells contain both heparin and histamine.[29] Their function in the body has not yet been defined.

Nucleic acids. The R.N.A. concentration is greatly increased in the basal cells of the marginal epithelium but decreased in all the crevicular cells. In any attempt to assess intensity of stains, even semi-quantitatively, care must be taken when interpreting the results. Cell size must be considered,

because a small cell staining deeply may contain the same amount of a substance as a larger cell staining less intensely. However the deeper staining of R.N.A. did not seem entirely dependent on the size of the cell because, although the superficial cells were frequently slightly larger than the basal cells, the concentration of stain was many times lower.

The lower R.N.A. concentration in the crevicular epithelium may have been due rather to infiltration of the tissue by various elements than to any difference in chemical composition between crevicular and marginal epithelium because those regions of the marginal epithelium of inflamed gingivae infiltrated by leucocytes also showed lower R.N.A. content.

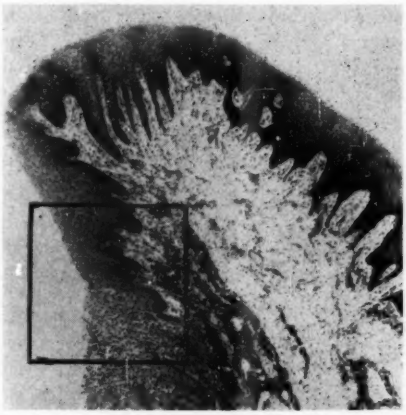

Fig. 5. Section from inflamed gingiva, stained to demonstrate nucleic acid content. Decreased ribonucleic acid in the lower crevicular epithelium. This zone is infiltrated by leucocytes (Toluidine blue: magnification approx. 35 X).

INFLAMED GINGIVAE

Keratin. A decrease in degree of keratinization occurred with inflammation of the gingiva, specimens from eight subjects being non-keratinized. In six of these eight subjects there were changes in the endocrine state. However many cases of puberty, pregnancy and diabetes did have keratinized epithelium.

Carbohydrates. Glycogen: The deposition (i.e. increase) of glycogen in inflamed gingival tissue has been noted by Ziskin, Loughlin and Siegel,[37] Engel, Ray and Orban[12] and Turesky, Glickman and Litwin[35] but no explanation has been given of the cause or mechanism of deposition.

On the other hand, Berg, Lundquist, Schram and Fosdick[1] found that a decrease in concentration of glycogen occurred with trauma or inflammation of the gingiva. A critical appraisal of the technique used reveals a possible explanation for these conflicting results. The method of estimation of the glycogen was one which has been proved specific for purified preparations of glycogen, but its reliability and specificity when applied to pieces of tissue, containing large quantities of glycoprotein, is open to doubt. The estimations were made on specimens which included both epithelium and connective tissue. In trauma and inflammation, alteration occurs in the glycoprotein content of the connective tissue and this may have influenced the results.

The *mechanism of deposition* of glycogen in epithelial cells of the gingival tissue can be explained on the basis of the work of Menkin,[24] who has shown that in inflammation in diabetics protein of the damaged tissue is broken down to glucose (gluconeogenesis) and there is an increase in glucose in these inflammatory exudates. In the inflammatory exudates of non-diabetics this glucose is usually removed by glycolysis, but all the glucose may not be broken down in inflamed gingiva. It may be that either the production of glucose is greatly in excess of that removed by glycolysis or that glycolysis in the gingiva is not as active as in exudates. This phenomenon of gluconeogenesis has not been considered at a cellular level but it could supply glucose for the glycogen found in the epithelium of inflamed gingiva. Although dyes and other complex molecules are

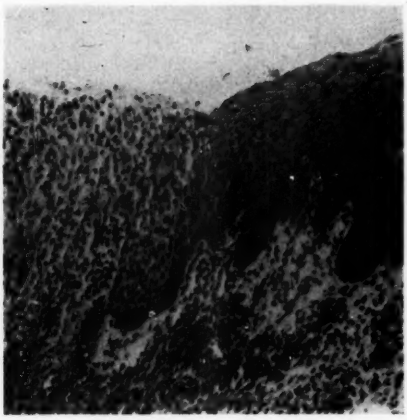

Fig. 6. Higher magnification of section shown in Figure 5. On the left decreased ribonucleic acid content of the cytoplasm of the zone of epithelium infiltrated by leucocytes (Toluidine blue: magnification approx. 100 X).

trapped in the sites of inflammation, it has been shown that glucose will diffuse out of such sites[25] and it could diffuse into the epithelium. The presence of glycogen tends to decrease proteolysis; however the histochemical evidence shows that the glycogen is not deposited in the zone where proteolysis is taking place and therefore it cannot exert a direct protective effect, as was suggested by Berg and co-workers[1] and Ziskin and co-workers.[37]

The highly polymerized carbohydrate of the ground substance of the connective tissue is diminished in inflamed tissue and glucose could also be derived from this substance. The exact composition of the ground substance is not known with certainty but it is usually considered to be a glycoprotein, which contains hexosamine and hexuronic acid but not hexose units; whether there is a deaminase capable of deaminating hexosamine is not known.

Engel et al.[12] noted a decrease in the ground substance of connective tissue and an increase in the water-soluble mucoprotein in cases of desquamative gingivitis. Although they drew attention to the collagenase of Clostridium welchii and the mucinase of Vibrio cholerae, which have the ability to break down glycoprotein, they consider that in desquamative gingivitis most of the changes in the ground substance may be due to a polymerizing enzyme released from the tissue by hormonal action.[15] In the inflamed gingivae examined in this work it is possible that the destruction of the glycoprotein could be due partly to bacterial enzymes. The gingival pocket has been reported to contain bacteria capable of producing hyaluronidase[23] and collagenase,[31] and hyaluronidase could facilitate penetration of the other enzymes through the crevicular epithelium.[32]

The pattern of deposition of glycogen, observed in this work to be remarkably constant (Figures 1 and 2), is partly explained by the work of Montagna, Chase and Lobitz[26] and of Dempsey and Wislocki.[9] Montagna and co-workers found that if the amount of glucose supplied to cells was large glycogen would be deposited in the older cells but not in the metabolically-active cells where growth and cell division were taking place. Dempsey and Wislocki found that glycogen was deposited in the metabolically-sluggish and poorly vascularized tissue. The concentration of R.N.A. is high where growth is taking place. In inflamed gingival tissue glycogen was not deposited where the R.N.A. concentration was high, that is, in the basal cells of the marginal epithelium —these cells were apparently using the glucose for growth and cell division. In the upper Malphigian and superficial layers R.N.A. concentration was low (Figure 4) and mitotic figures entirely absent—these cells apparently did not need the glucose for their metabolism but deposited it as glycogen.

In the healing of skin wounds[6] and in the cyclic changes in the vagina[5] and endometrium[9] glycogen deposition has been associated with keratin production, the

glycogen being considered a possible source of energy for the production of keratin in areas of lowered oxygen tension. Because the non-keratinized crevicular epithelium contained no glycogen deposits and the keratinized marginal epithelium frequently did, the possibility of an *association of glycogen deposits and keratin production* in the gingival tissue was investigated. It was originally considered that glycogen deposition might be solely a reserve for keratin production and that deposition would occur only in areas where keratinization takes place. However examination of the gingival tissue showed that glycogen was deposited without relation to the state of keratinization: glycogen deposits were seen in thirty specimens of keratinized marginal epithelium and in twenty-three specimens of non-keratinized epithelium. In normal gingivae similar zones were all keratinized and free of glycogen deposits. It was thought that in the non-keratinized areas another factor necessary for keratin production, e.g. physical stimulation, might be absent, so that the relationship between keratinization and glycogen deposition could not be determined in this tissue.

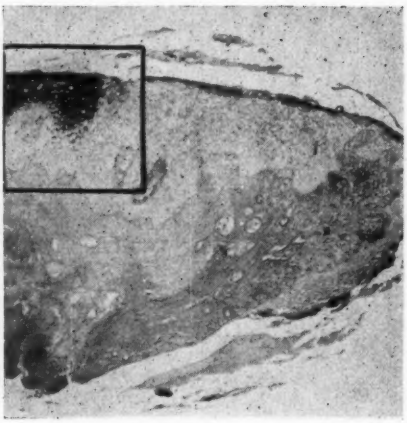

Fig. 7. Section from inflamed gingiva, stained to demonstrate glycogen. The crevicular epithelium and upper marginal epithelium are free of glycogen deposits. These zones are infiltrated by leucocytes (Hotchkiss stain: magnification approx. 35 X).

In areas where the epithelium was infiltrated by leucocytes and slight destruction of intercellular substance had occurred no glycogen was deposited (Figures 7 and 8). All crevicular epithelium examined was infiltrated in this way and this infiltration, rather than the absence as such of keratinization, possibly explains why no glycogen deposition occurs in the crevicular epithelium. When tissue was inflamed, heavy deposits of glycogen occurred in the marginal epithelium but when the inflammation reached the stage where leucocytes infiltrated into the epithelium, the glycogen deposits were not seen (Figures 7 and 8). A very different pattern was observed where part of the epithelium was infiltrated: the infiltrated area of the epithelium was free of glycogen, although heavy deposits occurred in the remainder of the epithelium. Hence glycogen may be formed in the tissue when it becomes inflamed but when the area of epithelium becomes infiltrated with leucocytes and other elements the glycogen is broken down; also glucose, both the normal supply and the excess, could diffuse out of the disorganized tissue and thus not remain in sufficient concentration to supply energy for keratinization or to allow deposition of glycogen.

Epithelium of the gingivae, whether marginal or crevicular, was free from R.N.A. as well as glycogen when infiltrated by leucocytes and other elements (Figures 6 and 8). Saliva contains both amylase and ribonuclease and these enzymes could possibly find entry into the disorganized tissue. However an investigation of ulcerated skin[10,13] showed decrease in the glycogen and R.N.A. of the epithelium, so that the changes in the gingiva may be due to infiltration of enzymes, derived not from the saliva but from the inflamed area. Leucocytes have been found to contain many enzymes, including amylase and nuclease but they were not entirely responsible for removal of R.N.A. and glycogen, because these compounds were not diminished in

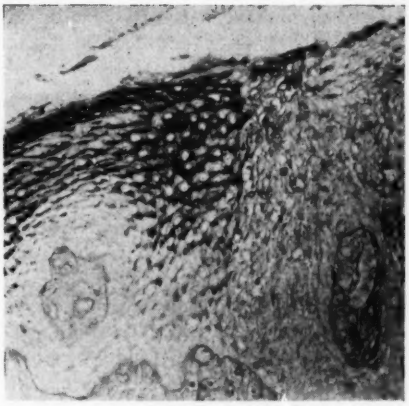

Fig. 8. Higher magnification of section shown in Figure 7. On the right the zone of epithelium is free of glycogen deposits. This zone is infiltrated by leucocytes (Hotchkiss stain: magnification approx. 100 X).

zones of the epithelium where leucocytes had infiltrated a few layers and had not reached the surface.

It is therefore suggested that deposition of glycogen in the inflamed gingivae is dependent on glucose supply—the presence of an excess of glucose (from gluconeogenesis) which diffuses into the epithelium. The glycogen deposited is apparently in excess of any required as a source of energy for keratinization and other syntheses and its deposition is simply a means of temporarily disposing of unwanted glucose.

PATHOGENESIS OF GINGIVITIS

Much has been written of the causes of gingivitis and the mechanism of inflammation of the gingiva. The one outstanding clinical observation is that, if the local irritant is removed from the gingival pockets, the gingivae heal.

The irritant was claimed by early investigators to be the bacteria present in the gingival pocket. Several investigators have shown that the exudates from periodontal pockets are "toxic" when injected subcutaneously although "non-toxic" when applied topically to the oral mucous membrane. Graham[17] and Rosebury[30] carried out tests on animals with material removed from the peridontal pocket, that is, bacteria, bacterial end-products and tissue breakdown products. When this material was injected severe necrotizing reactions usually followed. However bacteria have rarely been detected in inflamed gingival tissue. Fish,[14] Bibby[3,4] and Kimball[20] all suggest that bacterial products, and not the bacterial cells, enter the tissue and cause the inflammatory reaction. When skin tests were performed, the injection of exudates that were sterilized by filtration through sintered glass filters (which retain bacteria) caused reactions, although slight.[4]

In the present investigation no bacteria were detected in the tissue, except when a path of entry was apparent. It has been shown that hyaluronidase can facilitate such a penetration through the crevicular epithelium of the gingiva,[32] and that hyaluronidase and collagenase are present in the pockets of inflamed gingiva. Other relevant facts of interest are that protein degradation products, such as peptones, can cause mobilization of inflammatory cells[27] and that peptone[18] and histamine[34] can increase the spreading power of hyaluronidase.

It is known that proteolysis takes place in the periodontal pocket,[20] so that the leucocyte-attracting power of protein degradation products may have a role in the process of inflammation in the gingivae. It is suggested, therefore, that in the initiation of gingivitis hyaluronidase from bacteria in the gingival pocket allows penetration through the crevicular epithelium; the degradation products of proteins present in the gingival pocket then enter the gingiva through these zones of increased permeability and initiate or intensify the inflammatory reaction. Sometimes, as the disorganization of the crevicular epithelium progresses, the bacterial cells also penetrate

the tissue. The presence of large numbers of plasma cells in the chronically inflamed gingival tissue is of interest, because these cells are thought to produce and liberate antibodies which neutralize injurious antigens, although evidence is largely circumstantial.[7] The presence of plasma cells may perhaps be taken as an indication of the presence of antigenic foreign protein and peptones in the tissue. Systemic disturbances, particularly hormonal, with resultant changes in tissue permeability, affect the ease of spread of the irritating agents and the progress of the inflammatory reaction.

SUMMARY AND CONCLUSIONS

1. Tissue from the gingivae of fifty-six patients was studied. Eight patients had clinically normal gingivae, seven were suffering from slight marginal gingivitis and forty-one from gingivitis or periodontitis.

2. Histochemical methods were utilized for the demonstration of glycogen, glycoprotein, mucopolysaccharides, keratin, alkaline phosphatase and nucleic acids.

3. Glycogen deposits were observed in the marginal epithelium of inflamed gingivae. An hypothesis is advanced that the glycogen is formed from excess glucose produced in the subjacent inflamed tissue by gluconeogenesis. The older cells of the marginal epithelium deposit the glucose as glycogen but the younger, more active, basal cells use it in growth and proliferation.

4. Glycoprotein of the ground substance was diminished in zones of inflammation. Very little glycoprotein was observed in the intercellular substance in the basal layers of the marginal epithelium or in the entire crevicular epithelium of both clinically normal and inflamed gingivae.

5. Mast cells were found in large numbers in the gingiva. Their number was increased significantly when the gingiva was inflamed.

6. Thirty-five of forty-one specimens of inflamed gingiva showed keratinized marginal epithelium. No crevicular epithelium was keratinized, whether the gingiva was clinically normal or inflamed. *All* crevicular epithelium was infiltrated by leucocytes, with some destruction of intercellular substance. Lack of glycogen deposition in non-keratinized areas, such as the crevicular epithelium, was thought to be due to infiltration of the tissue by leucocytes and other elements and possible diffusion of glucose out of the tissue and not to lack of keratinization.

7. Alkaline phosphatase was present in the nuclei of endothelial cells lining the vessel walls and in the nuclei of inflammatory cells.

8. The D.N.A. content of the tissue was constant. The R.N.A. was greater in the cytoplasm of the basal cells of the epithelium than in the older cells of the superficial layers. The crevicular epithelium contained only a small quantity of R.N.A. Infiltration of the gingival epithelium by leucocytes and other elements lowered the R.N.A. content of the infiltrated zone.

9. A pathogenesis for gingivitis is discussed.

ACKNOWLEDGMENTS

I wish to thank Mr. G. Hutchinson, Department of Periodontia, United Dental Hospital of Sydney, for collecting tissue, also Mr. E. Slater, Department of Photography, Faculty of Dentistry, University of Sydney, for preparing the photomicrographs.

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Editorials

IT'S EASIER TO BE NEGATIVE

In the December 1954 issue of *The Journal of the American Dental Association* can be found an informative editorial entitled "Some Advice on Giving Advice on Nutrition."

The editor writes: "A report of the Council on Dental Therapeutics of the Association on certain aspects of nutrition to be included in the 1955 edition of *Accepted Dental Remedies* might well become a permanent part of every dentist's library as it cautions members, by implication, against becoming involved with those pseudoscientists whose nutritional ministrations consist largely of a mixture of old wives' tales and empiricism. Ever since man first became conscious of the relation between food and his physical being, countless crimes have been committed in the name of nutrition. . . .

"What the field of nutrition holds in store for man in the future, only the future can tell. But it is hoped for the well being of the generation of tomorrow, that the empiricism of the food faddist soon will give way entirely to the research of the scientist.

"Every nutritionist knows that good nutrition depends on many things other than the intake of an adequate diet. Many of those factors remain unknown, yet the commercial food faddist ignores these blanks in nutritional knowledge and by means of radio, television, newspaper and magazine would have listeners believe that the product which he promotes is the true elixir of life. Because many vendors of vitamins are particularly obnoxious in this respect and because some members of the dental profession, unfortunately, are unduly influenced by their claims, certain precautions . . . are in order.

"Unless the dentist keeps himself well informed on the latest aspects of nutrition, he would be wise to speak sparingly on the subject lest he unwittingly align himself with that endless group who blithely continue to err in the interest of nutrition.

"It also behooves the dental schools to insist that their graduates are not only well grounded in the latest scientific knowledge of nutrition but also imbued with a healthy skepticism regarding claims and theories relating to those facts of the field where research has not yet yielded substantial results."*

We approve heartily of this editorial and publish a summary of it to emphasize the importance of being scientific in our discussions of nutrition with patients. However, the editorial is negative in tone. There is need for *positive* statements which emphasize what should be done for patients. Unfortunately, such information is not too readily available and what is is often not too

*J.A.D.A. 49:718 (Dec.) 1954.

well substantiated. Recognizing the importance of nutrition and the desirability of emphasizing it to patients, the editorial staff of the Journal actively solicits good articles on nutrition which state how to determine the nutritional status of patients, and give dietary instructions for them to follow.

NEED FOR NATURAL BRISTLES CITED

A peculiar situation has arisen regarding the supply of natural bristles for the manufacture of toothbrushes. Since the Korean conflict the supply of top quality bristle has been cut off, since apparently the best bristles come from China. Manufacturers report that the supply of natural bristle is so curtailed that the supply of natural bristle toothbrushes is threatened. In Miami on November 6, 1954, the Executive Council of the American Academy of Periodontology considered this problem and finally adopted the following resolution:

"WHEREAS, There are many dentists who definitely prefer and prescribe natural bristled toothbrushes in various designs and textures and,

"WHEREAS, There is a relationship between the public dental health and a supply of toothbrushes with natural bristles and,

"WHEREAS, We, as an organization especially interested in the health of the supporting tissues of the teeth, believe that in the field of preventive dentistry, there is no type of natural bristle superior to that of China origin.

"BE IT RESOLVED, That this Academy petition the Office of Defense Mobilization to release from the government stockpile an adequate supply of natural hog bristle for toothbrushes in the interest of dental health."

Also in New York late in December the brush manufacturers of America discussed this problem at a meeting in which most toothbrush manufacturers were represented. They finally recommended also that a small amount of the bristle which has been stockpiled by the United States Government be released to them to insure a continued supply of natural bristle brushes.

A discussion of whether or not natural bristle brushes are superior to those made of nylon filament is beyond the scope of this editorial. Suffice it to say there are many dentists who firmly believe natural bristles to be superior and since only a small amount of natural bristle is required to maintain this supply we believe everything should be done to convince the governmental agencies that natural bristles should be released to the brush manufacturers.

REGARDING MEMBERSHIP

Membership in the American Academy of Periodontology has become such a coveted honor that the Membership Committee has been hard pressed in studying the qualifications of the many applicants. Dr. Hunter S. Allen, Chairman of the Membership Committee, asks that the following notice be published and that

all members proposing names for consideration for membership read these regulations:

1. According to the Constitution and By-Laws, one cannot apply for membership. It is by invitation only, following recommendations by the Membership Committee and the Academy Council, and election by the general membership.

2. Proposals and/or seconding for membership in the Academy may be made by *Active* members only.

3. It is requested that no more than two proposals or secondings a year be made by an Active member.

4. A dentist who is interested in, and devotes part of his practice to periodontia, may be proposed as an Associate member five or more years after his graduation from a dental school.

5. An Associate member who has demonstrated the necessary proper qualifications and an active interest in the affairs of the Academy, may be proposed for Active membership after five years, or earlier through special recommendation by the Council.

6. Proposals for membership in the Academy must reach the Secretary of the Academy by January 1st, for consideration during that year.

MIAMI MEETING SUCCESSFUL

The 38th Annual Meeting of the American Academy of Periodontology which was held in the Empress Hotel in Miami, Florida, was outstanding judged by any standard—quality of program, size of attendance, caliber of audience, success of official functions. It is most gratifying to the "old timers" in the Academy to realize that this organization has developed until now there is never any doubt about the importance of each annual meeting.

Th editor had hoped and planned to have a rather detailed report of the Miami Meeting for publication in this issue of the Journal. Several reports are still now available, however, so after waiting until ten days beyond the "deadline" we decided to publish the report in April.

Oral Hygiene Habits of Young Adults*

by LILLIAN O. DAHL AND JOSEPH C. MUHLER, *Bloomington, Indiana*

IN a previous study from these laboratories,[1] the oral hygiene habits of children was presented. It was shown in a group of 514 children, age 6 to 15 years, that the most frequent time of brushing the teeth was before breakfast and before retiring. The frequency of brushing for the entire group was 1.4 times per day with the required time to brush the teeth being one minute for boys and 70 seconds for the girls. Less than 5 per cent of the children brushed their teeth three times a day in accordance with the suggested recommendation of the dental profession. Since it has been recently suggested that a synergism exists between good brushing habits and the anti-cariogenic effects of a stannous fluoride dentifrice[2] it was considered of general interest to study the oral hygiene habits of a group of young adults in order to determine if an increase in oral hygiene occurs with increasing age, as well as to learn more about the toothbrushing habits of these adults which have shown a reduction in the dental caries experience through the use of a stannous fluoride dentifrice.

EXPERIMENTAL PROCEDURE

In this study a total of 340 Indiana University freshmen, ages 17 to 34 years, was used. The mean age of the group was 19 years. These students were selected at random from the 1952 freshman class of Indiana University, with an attempt to maintain equal distribution of sexes. The entire group entered the study program throughout a period of three months and were processed in a manner similar to that described previously for the children's study.[1] Briefly, this consisted of the patients initially coming to the Dental Clinic for a thorough dental prophylaxis and dental caries examination including a complete bite-wing radiographic examination. After a period of six weeks they returned to the clinic and were questioned regarding their oral hygiene habits. At this time they were provided with a new toothbrush and dentifrice and asked to brush their teeth in the same manner as they would at home. Following this their mouth was re-examined and their brushing efficiency evaluated. Additional items used for evaluation in this study were also recorded during this visit. Six months following the first prophylaxis in the program they returned for another dental prophylaxis, oral and radiographic examination and were questioned regarding other factors relating to their oral hygiene. One important factor considered at this time was an evaluation of the rate of calculus formation.

At six-month intervals following the initial visit a similar routine regarding prophylaxis, oral and radiographic examination was followed. However, these data presented in this paper concern only the pertinent findings at the end of one year.

DATA AND DISCUSSION

These data collected from this study are summarized in tables 1 through 6. Table 1 shows the time of day the university freshmen brushed their teeth, and indicates that the largest single group of students brush before breakfast and before bed. This comprises 21 per cent of the total group and corroborates the findings observed in the children's study.[1] About 11 per cent of the total group brushed their teeth with a frequency of three times a day after meals. This is approximately 50 per cent greater than similar comparisons in the younger age group. It appears from these limited data that young adults are more conscientious in their efforts to brush their teeth imme-

*Indiana University, School of Dentistry. This study was supported in part by a grant from the Procter and Gamble Company, Ivorydale, Ohio.

TABLE 1

The Time of Day 340 College Students, Participating in a Dental Survey, Brushed Their Teeth

Time of Day	*Number of Students*	*Percentage*
Before breakfast	26	7.6
After breakfast	13	3.8
Before lunch	0	——
After lunch	0	——
Before dinner	0	——
After dinner	1	0.3
After breakfast and after dinner	19	5.6
Before breakfast and after dinner	15	4.4
After breakfast, lunch and dinner	39	11.5
After breakfast and before bed	42	12.4
Before breakfast and before bed	70	20.5
After breakfast, lunch and dinner and before bed	23	6.8
After breakfast, after lunch and before bed	12	3.5
Before bed	21	6.2
Irregular	59	17.4

diately after the ingestion of food than are children. A somewhat larger portion of the group, 17 per cent, were found to be irregular in their brushing habits and were not able to be classified in any of the divisions indicated in table 1. These data, combined with those in table 2, corroborate the fact that the frequency of brushing per day is below that figure considered optimal for good dental health, since the average for both the male and female groups is only slightly in excess of twice a day. The males have a mean of 1.7 times a day and the females 2.1 times. Thus, it appears that the females in the study, as in the children's study,[1] are more conscientious in their brushing habits. Also, it is evident that this young adult group has increased their frequency of brushing per day over the group participating in the children's study by approximately 30 per cent. However, the data in table 3 seems to indicate that the time required to brush the teeth of the females is somewhat less than that for the males. This is observed in both the total time required as well as the range of time needed to brush the teeth. The males brushed on the average of 64 seconds and the females 57 seconds, with the range for the former 15 to 135 seconds and 15 to 117

TABLE 2

Number of Times Per Day 340 College Students Brush Their Teeth

	Times per day							
Group	*1*	*2*	*3*	*4*	*5*	*6*	*irregular*	*average*
Male (n = 187)	69	75	32	6	1	0	3	1.7
Female (n = 153)	27	69	28	14	9	1	0	2.1

TABLE 3

Length of Time Required by Young Adults to Brush Their Teeth

	Mean number of seconds brushed	*Minimum (seconds)*	*Maximum (seconds)*
Male	63.5	15	135
Female	56.7	15	117

seconds for the latter. Further evidence for this increased time requirement for males to brush their teeth is shown in table 4, where the percentage of individuals in each brushing time interval is shown. These data indicate that about 5 percent of the males and 8 percent of the females require less than 30 seconds to brush their teeth. However, about 50 per cent of both sexes fall into the 30 to 60 second range. From these data it may be seen that a greater number of females fall into the low brushing time range, while the males seem to have a tendency to brush for longer periods. These findings, in regard to the length of time required to brush the teeth, are not significantly different from those data found in the younger age group.[1]

Table 5 indicates the condition of the oral hygiene at the initial visit and again after six months following the initial dental prophylaxis. These data are somewhat difficult to interpret in light of the data found in tables 2 through 4, since it appears from the data in table 5 that the females possess a better state of oral hygiene. Approximately 50 percent of the females were classified initially as "very good" or "good" (52.3 percent) while less than 40 percent of the males were similarly classified. After six months in the study program the same conclusions are evident. About 4 percent of the males were classified as having "very poor" oral hygiene at the initial visit while 2.6 percent of the females were similarly classified. The greatest change in the condition of the oral hygiene during the six month period occurred in the male group classified as "poor". Initially, approximately 16 percent were classified as "poor" but after six months less than 5 percent were included in this category.

Some estimate of the rate of calculus formation in this young adult group can be seen from the data comprising table 6. Initially, 67.9 percent of the group were essentially free of dental calculus, while 11 percent were classified as having a severe amount. Following a thorough prophylaxis and a period of six months, 5.7 percent were classified as severe. The decrease in the severe group was not shared by an increase in the calculus free group, but was apparent by an increase in the slight and medium

TABLE 4

Range of Time Required to Brush the Teeth

Group	*Time (in seconds)* 0-30		30-60		60-90		90-120		120 or over	
	No.	%	No.	%	No.	%	No.	%	No.	%
Male	5	4.9	49	47.5	32	31.1	14	13.6	3	2.9
Female	8	8.2	54	55.7	28	29.9	7	7.2	0	—

TABLE 5

The Condition of Oral Hygiene in College Students at Initial Visit to the Dental Clinic and Six Months Later in the Same Patients

	Very Good		*Good*		*Fair*		*Poor*		*Very Poor*	
					Males					
	No.	%	No.	%	No.	%	No.	%	No.	%
Initial Examination	17	9.1	54	28.9	80	42.8	29	15.5	7	3.7
Six Month Examination	18	9.6	66	35.3	91	48.7	9	4.8	3	1.6
					Females					
Initial Examination	18	11.8	62	40.5	56	36.6	13	8.5	4	2.6
Six Month Examination	19	12.4	64	41.8	57	37.3	13	8.5	0	—

groups. These data appear interesting in light of the fact that a much larger percentage of the population in the age group over 35 falls into the severe classification. Thus, it appears that in this younger age group calculus formation is not a rapid process.

SUMMARY

The oral hygiene and toothbrushing habits of a group of 340 freshmen students at Indiana University were studied for a period of one year in order to ascertain pertinent facts relating to the oral hygiene. It was apparent from the data in this study that the toothbrushing habits in young adults are quite similar to those of a children's group. The single factor which is different in the adult study is in relation to an increase by approximately 30 percent in the frequency per day of toothbrushing. There appears to be little relationship between the time required to brush the teeth and the overall oral hygiene, since females brush for shorter periods of time but more are found to have a good oral hygiene than males. Only 11.5 percent of this group brushed their teeth three times a day after meals.

The rate of calculus formation was estimated during a six month period following a thorough dental prophylaxis. It was observed to be a very slow process in this young adult group. More study is indicated concerning this latter phenomenon.

TABLE 6

Calculus Formation in a Young Adult Group During a Six Month Study Period

Amount of Calculus	*Initial Examination*		*Six Month Examination*	
	Number	*Percent*	*Number*	*Percent*
None	191	67.9	188	66.9
Slight	46	16.4	53	18.8
Medium	13	4.6	24	8.5
Severe	31	11.0	16	5.7

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The Psychosomatic Aspect of Periodontal Disease

by CYRIL B. KANTERMAN, D.D.S., *Uniontown, Pennsylvania*

BY definition, psychosomatic medicine is the study dealing with the interrelationship of emotional disturbances with pathologic structural changes. Psychosomatic dentistry, therefore, refers to the relationship between mental health and the health of the oral and dental tissues.[1]

The psychosomatic factor has long been recognized by the physician as an important factor in the etiology and treatment of disease. The dental practitioner has likewise come to accept the concept that emotional stresses may, at times, create oral problems, or at least be a contributing etiologic factor.

That periodontal disease may have, in part at least, a psychogenic basis is a view now generally accepted by the periodontist.[2] In the course of everyday living, the individual is continually beset with emotional conflicts and tensions of varying types and degrees. The so-called well-adjusted individual is able to resolve his emotional difficulties and to find an adequate outlet for these tensions. However, when an outlet cannot be found by word or action, the body will find a means of expressing this through some physical or somatic manifestation.[3] The oral tissues are good indicators of the general health of the body[4] so it is not surprising that these same tissues are likely to be among the first to be affected when the individual undergoes emotional stress.

While the connection between oral disease and emotional imbalance is not always readily apparent, many oral pathologic conditions appear to have a definite psychosomatic background. Lichen planus is a good example of this. Patients with this disease are usually emotionally hyperactive, worrying individuals who invariably have a fear of cancer.[5] Roth[6] cites a case of recurrent ulcerative necrotizing gingivitis in which a psychosomatic factor was predominant. Investigations conducted by Mellers and Herms indicate that patients who have emotional problems of any type are, in the majority, subject to gingival changes resulting in bleeding gingival tissues and recession of the tooth-supporting structures.[7, 8] The astute diagnostician, whether he be a periodontist or general practitioner, recognizes the possibility of a psycho-pathologic relationship, and is always on the lookout for an emotional factor in the formulation of his diagnosis.

ASPECTS OF THE PROBLEM

There are two broad aspects to the problem of psychosomatics in the field of periodontal disease. First of all, emotional disturbances may affect certain physiological processes or functions of the body in such a way that the oral tissues are directly and

adversely affected. An example of this may be cited in the fact that emotional stress has been known to cause a marked increase in salivation in many individuals. It can readily be seen that if this psychic tension is allowed to continue, the amount of saliva secreted in the oral cavity will be at an abnormally high level. This, in turn, will permit the rapid formation of salivary calculus, thus giving rise to a possible etiologic factor in periodontal disease.[9]

Another example of how the emotions may affect the physiology of the body as it relates to the periodontal tissues is shown in the fact that emotional stress has been reported to cause a change in the chemical composition of the saliva. Briggs[10] calls attention to the fact that adverse mental stimuli such as fear, hate, and uncertainty will decrease the calcium content of the saliva, while tranquillity and peace of mind will tend to increase it. Other investigators have shown a positive correlation between the acidity of the saliva and increased emotional tension.[11] When many individuals are faced with a conflict situation, the acid level of the saliva shows an abnormal increase due to autonomic stimulation of the salivary glands. The resulting hyperacidity in the oral cavity will not only have a deleterious effect upon the enamel of the teeth, but it will also permit the growth of certain microorganisms that are known to be causative agents in periodontal disease. Thus, a chemical change in the buffer state of the saliva, brought about by chronic emotional tension, may lower the threshold for attack by microorganisms in the oral cavity.[12]

Thus far, only one aspect of the problem has been considered, namely, the effect of the patient's mental and emotional state on the health of the oral tissues. The second aspect which must be considered is the reverse effect—the effect of the periodontal and other oral conditions on the emotional stability of the individual.

It is well-known that such disfiguring defects as malocclusion, harelip, or cleft-palate can cause considerable mental anguish to the individual to the point where even suicide may be contemplated.[13] The same undoubtedly holds true for less serious periodontal conditions, although perhaps not to such an extreme. Consider, for example, the psychological effect upon a patient of a rather common oral disease such as acute ulcerative necrotizing gingivitis. The symptoms accompanying this disease, such as sloughing of the tissues, gingival bleeding, and foul odor, would foster the feeling in the patient that his mouth is "dirty," thus tending to make him self-conscious and retiring. It is not inconceivable that an introverted personality can develop as a result of an oral problem.

PSYCHOSOMATIC MECHANISMS

The question may now be raised: In what specific ways can psychosomatic factors initiate periodontal disease? Miller[14] lists a number of mechanisms by which this may occur:

1. *By reduction of local nutrition through vascular changes.*

Chronic emotional disturbances, such as excitement and depression, may cause vascular changes in the periodontal tissues, giving rise to a pathological condition in these tissues. Angioneurotic edema, for example, may be precipitated emotionally.[15] Chavoor[16] points out that chronic tension may set up a vasoconstriction of the minute capillaries supplying the mucous membrane and gingival tissues, so that there is interference with nourishment to the cells. In time, the localized anoxia may so lower tissue resistance that bacterial invasion easily occurs. Other investigators have shown that vasal stimulation causes dilation of the capillaries, resulting in hemorrhage and hypertrophy of the tissues, while vasal depression leads to constriction of the capillaries, producing atrophic changes in the supporting gingival and alveolar tissues due to defective nutrition in these tissues.[17]

2. *By increased bacterial flora.*

In a state of depression, the individual is likely to pay less attention to his health or to his habits of hygiene, so that the hygienic condition of his oral cavity is likely to be poor. It has been shown that there was a marked increase in the causative microorganisms of Vincent's infection during a period of emotional stress, and that there was a significant decrease in these same microorganisms when the emotional disturbance was eliminated.[18]

3. *By the development of habits, both objective and subjective, which are harmful to the health of the periodontal structures.*

There are quite a number of objectionable oral habits, that if practiced continually, will give rise to injury to both the hard and soft tissues of the oral cavity. Many of these habits are psychogenic in nature and are performed unconsciously by the individual. They include lip biting, cheek biting, toothpick biting, abnormal occlusal habit resulting from nervousness, occlusal and incisal grinding, clenching of teeth in the control of emotions, fingernail biting, and abnormal tongue pressure against the teeth.[19]

4. *By inducing excessive chewing, clenching, or grinding.*

These habits create not only marked abrasion of the teeth, but also excessive pressures that result in injury to the supporting tissues. One of the most common of such pernicious habits is bruxism, which is the grinding of the teeth during sleep. Miller[20] maintains that in every case of bruxism the core of the problem is to be found in the personality development of the individual. Thus, it would seem that bruxism is primarily an expression of the psychological make-up of the individual, of which his personality is a part. Ingersoll and Kerens[21] state that in many of the cases of bruxism a mild form of neurasthenia may be present.

5. *By creating taste perversions causing the ingestion of foods which are locally and systemically harmful to periodontal health.*

When under emotional tension, many individuals sometimes develop an abnormal desire for certain types of foods. These include excessive quantities of candy, pastries, soft drinks, and alcoholic beverages. Others may develop appetites for citrus fruits or highly spiced foods. All of these foods have a direct traumatic effect upon both the teeth and supporting periodontal tissues. Also in this category, excessive smoking may well be considered as having a deleterious effect on the soft tissues of the oral cavity.[22] The so-called chain-smoker is invariably a chronically hypertensive individual, or one who is under considerable mental or emotional stress.

6. *By permitting insufficient food intake through limitation of gastrointestinal function.*

There are a number of gastrointestinal disorders that are essentially psychogenic in nature.[23] These include gastrospasm, esophagospasm, gastric and duodenal ulcers, anorexia nervosa, and colitis. Any one of these conditions, if present in the individual, will limit the quantity of food intake due to an insufficiency in gastrointestinal function. There is not sufficient food ingested to properly maintain body health, with the result that the periodontal tissues as well as other tissues in the body are likely to suffer. Thus, indirectly a psychogenic disease may affect the health of the periodontal tissues.

7. *By producing neglect of oral sanitation.*

Individuals who are mentally occupied or depressed will tend to neglect proper oral hygiene. It has been shown that such individuals tend to accumulate calculus more rapidly than do other types of patients.[24]

8. *By causing bodily conditions inimical to the health of the periodontal tissues.*

Certain physiological changes occur in the emotionally unstable individual that have a direct effect upon the periodontal tissues. Investigators[25] have found that a relative decrease of blood calcium and phosphorus occurs in tense and agitated patients. Likewise, a low sugar tolerance and glycosuria are common findings in emotionally upset individuals.[26] These systemic physiologic changes, in turn, adversely affect the well-being of the oral tissues.

CONCLUSION

The psyche, when subjected to emotional conflicts over long periods becomes an important factor in causing a chain of somatic reactions that results in pathologic changes. Such changes affect neural, vascular, and endocrine systems which play a very important role in maintaining the health of the oral tissues.[27] Thus, it is apparent that psychosomatics play a definite and direct role in the etiology as well as the treatment of periodontal disease. It has been estimated that at least eighty per cent of dental patients have neuroses of some degree and complain of symptoms that may confuse the dentist in making a diagnosis.[28] From this fact it may be surmised that the psychosomatic factor perhaps plays a greater role in oral problems than has hitherto been supposed.

The importance of considering the patient's emotional needs is best summarized in the statement by Raginsky[29]:

"A dentist or a physician will never reach the optimal results in his own field regardless of his diagnostic acumen or his manual dexterity and skill if he does not take into consideration the patient's emotional needs. The ability to understand and handle a patient's emotional needs extends the scope and usefulness of the doctor."

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Idiopathic Hyperplasia of Gingivae Associated with Macrocheilia and Ankyloglossia: A Case Report

by ROBERT RAPP, GORDON NIKIFORUK, D. W. DONOHUE AND C. H. M. WILLIAMS,

Toronto, Canada

THIS case report covers a ten-year history of idiopathic hyperplasia of the gingivae in a boy sixteen years old.

The literature pertaining to gingival enlargements has been reviewed in the proceedings of the Periodontal Workshop.[10]

MEDICAL HISTORY

The earliest available medical record of this patient when he was six years old, revealed a history of swelling of the gums of at least two years' duration (Figure 1). Associated with the gingival condition were otitis media, sub-acute tonsillitis and carious teeth. Allergy tests revealed sensitivity to the following: Bananas, house dust, spinach and peas. Bacteriological smears showed a moderate number of fusiform bacilli and occasional spirochaetae. The recorded treatment during a two month interval was as follows: Topical application of Bowman's solution,* antibiotic lozenges containing sulphadiazine, penicillin, and streptomycin, and daily administration of ascorbic acid (100 mgs.). There was no other treatment and no further contact with the patient until July, 1953, when the patient was readmitted for investigation and treatment of the gingival condition.

The physical examination revealed a moderately well-developed healthy boy with enlargement of gingival tissue in both arches and an enlarged everted lower lip. The oral condition is illustrated in Figure 2.

Laboratory investigation revealed the following: urinalysis—no pathological constituents, hemoglobin 13.2 gms., white blood cell 12.4, tubercular and Wassermann tests—negative, blood culture—negative, fasting ascorbic acid level within normal limits, smear and differential counts normal. Allergy tests indicated strong sensitivity to cat and dog hair, june grass, orris root and a weak reaction to penicillium notatun and trichophyton. The urinary excretion of mercury in 24 hours was found to be grossly increased.

The family history gave no additional information about allergies. There was no rheumatic fever or nephritis in the family. The father is a controlled diabetic. Pregnancy and labor were normal.

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*Tincture of ipecacuanha and arsenical solution.

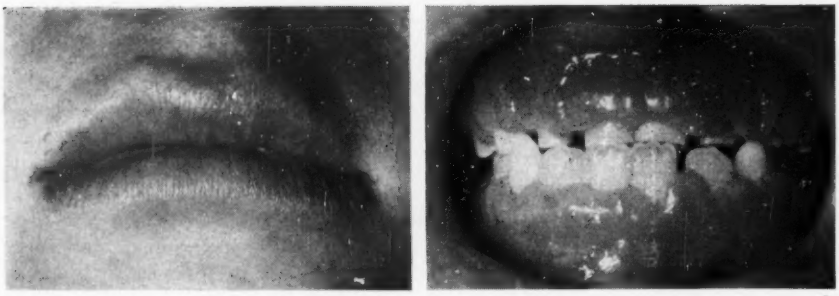

Fig. 1. Appearance of patient at six years of age. Left: Appearance of the lips. Right: The extent of gingival hypertrophy. (Photographs for Figure 1—courtesy of Dr. H. A. Hunter).

DENTAL HISTORY

The lower lip was grossly enlarged with a midline fissure of the mucous membrane. There was a generalized enlargement of the gingival tissue to an extent that some teeth were completely obscured (Figure 2B). This enlargement did not extend past the gingival tissue into the areolar mucosa. The tissue was bright red in color, firm, dense, resilient, and had lost its stippled appearance. Bleeding resulted from trauma such as would occur during brushing the teeth. Oral hygiene was extremely poor. Clinically signs of inflammation were present, but were not the dominant feature. The tongue was so restricted by the median frenum that its tip could not be protruded beyond the incisor teeth (Figure 3). Radiographic examination of all teeth revealed the presence of carious lower first permanent molars with rarefying osteitis about the root ends (Figure 4). There was a generalized loss of alveolar bone and a somewhat greater loss in the incisor regions.

HISTOLOGIC REPORT

Numerous sections were made of the tissue removed at the gingivectomy. All presented the same appearance. The tissue is in part covered by non-keratinized stratified squamous epithelium. Extending deep below the epithelium there is an intense inflammatory reaction composed chiefly of tremendous numbers of plasma cells with an occasional eosinophile (Figure

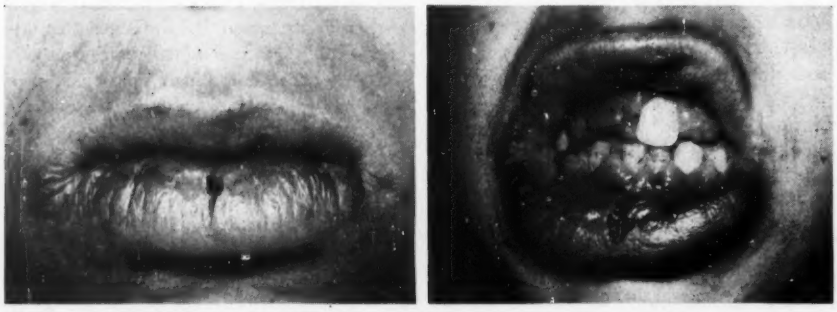

Fig. 2. The illustrations were taken when patient was sixteen years old. Left: Note the mandibular macrochelia that developed during a ten year interval. Right: Shows extensive gingival enlargement in both arches.

Fig. 3. Illustration of the ankyloglossia.

5A). There is also some fibrosis but this is not a prominent feature. In addition, there are a few pseudo-tubercle-like areas with proliferation of epithelioid cells and an occasional multinucleated cell but no caseation (Figure 5B). These structures are somewhat reminiscent of sarcoid. A battery of stains revealed no additional features and no organisms.

There is little doubt that the gross picture of this boy's gums and lip are similar to the picture described in the literature under fibrous hyperplasia. Nevertheless in this case the microscopic picture appears to be different, in that although some fibrosis is present the predominating feature is that of chronic inflammation.

ETIOLOGY

The basic factors involved in the etiology of this condition are unknown. Murray (1873) reported three cases of hereditary fibrous hyperplasia of the gingivae in one family; and Weski (1920) states that among 39 members in five generations of one family, 7 men and 9 women were afflicted by the condition. Savara et al. (1954) reported a case history where 6 siblings and a father were afflicted with hereditary gingival-fibrosis. The mode of inheritance in this family was apparently dominant. Endocrine dysfunction was suggested as a possible etiological factor.

In the case being reported no suggestion of a hereditary influence could be found. The influence of congenital factors could not be properly assessed. The routine laboratory findings were within normal values. However, the possibility of endocrine malfunction still remains. Anti-convulsive drug therapy such as sodium dilantin was ruled out as a factor in this case. Abundant calculus formation, carious teeth, and malocclusion were considered local contributing factors only. The great number of plasma cells in the microscopic picture suggests an antigen-antibody reaction. In view of the strong allergic history given by this boy one cannot discount the possibility of repeated contact with some material to which the boy has been sensitized,

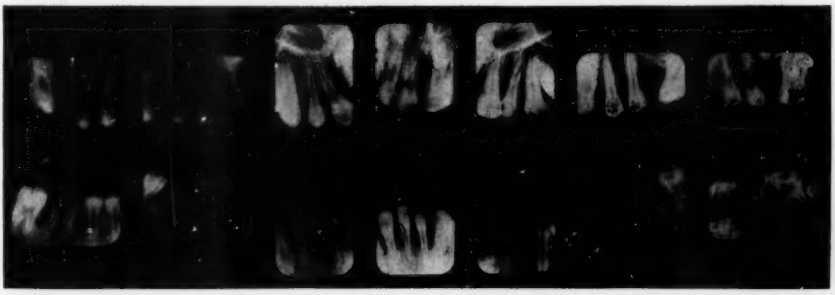

Fig. 4. Radiographs of the dentition. Note extreme loss of alveolar bone support, particularly in the mandibular incisor area. Areas of apical rarefaction around the lower first permanent molars were present.

Fig. 5A. Low power microscopic picture of a section of the gingivae.

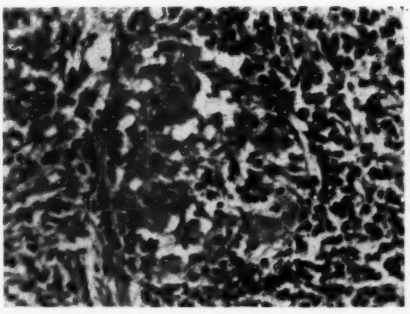

Fig. 5B. High power magnification of the area marked off in 5A. Note the marked inflammatory reaction and the presence of tubercle-like areas.

although careful investigation revealed no such mechanism.

In most of the cases reported other effects were noted. Feeble-mindedness, epilepsy, stunted growth, hypertrichosis, have been described as associated factors by Humphrey (1886) and Sarnat (1945). The present case was associated with macrochelia and ankyloglossia. The macrochelia increased when cold food or drinks were consumed suggesting a possible lymphatic stasis.

TREATMENT

The initial phase of treatment included extraction of the abscessed teeth and restoration of the carious teeth. An attempt was made to improve the oral hygiene but the condition of the gingival tissues prevented any noticeable success.

Gingivectomy was performed under local anesthesia, the four quadrants being treated within ten days. During each phase the patient received 400,000 I.U. of penicillin intramuscularly. The incised area was covered with germicidal periodontal pack

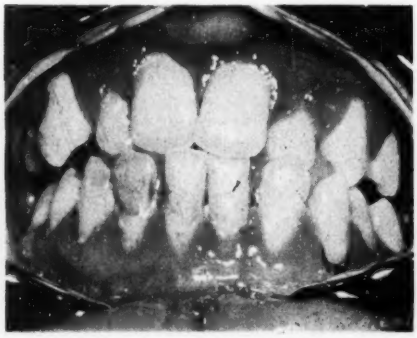

Fig. 6. Appearance of the case eight months after gingivectomy. Left: The lip has improved (compare with Figure 2, left). Right: There are definite signs of recurrence of the enlargement of the gingivae in the interproximal areas.

and the patient placed on a puree diet during the time the pack was in place.

After gingivectomy, scaling and polishing were completed. Any tags of granulation tissue that remained were removed with a saturated solution of trichloracetic acid.

The patient returned approximately five months after the gingivectomy. The lower lip was less enlarged than prior to the gingivectomy. There were signs of enlargement of the gingival tissue in the upper anterior region. This area also appeared highly inflamed with no stippling (Figure 6).

Ball (1944), Perkoff (1929), Hine (1952) and Newby (1940), have reported recurrence of the gingival enlargement after gingivectomy in similar cases.

CONCLUSION AND SUMMARY

A case of idiopathic hyperplasia of the gingivae in a boy sixteen years old extending over a ten year period is reported.

The medical and dental history do not reveal specific etiological factors in this case. The great number of plasma cells in the microscopic picture suggests an antigen-antibody reaction. The importance of the associated factors of macrocheilia and ankyloglossia have not been determined. Gingivectomy was at best a temporary palliative measure. There has been some recurrence of enlargement of the gingival tissue.

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Temporomandibular Joint Pathology Caused by Malocclusion and Premature Contact

by MAX SILVERMAN, D.D.S. AND HAROLD STANGEL, M.D., D.D.S., *Pittsburgh, Pa.*

THE temporomandibular joint, its pathological syndrome and contributory factor to facial neuralgia is becoming more and more the concern of the dental profession. This is evident by numerous articles on the subject in the dental literature, also by the discussion of the subject at the various dental seminars. It is a medico-dental problem which has needed attention for a long time.

Credit is due to the periodontists for bringing the attention of the dental profession to this ailment. In his daily contact with the periodontal patient, the periodontist observes the damage done to the temporomandibular joint by premature contact and malocclusion of the teeth. In many cases the subjective symptoms of pain and other manifestations of temporomandibular joint discomforts may be mild, but accumulative action, soon brings the problem to the dentist's attention.

Where malocclusion and premature contact of the occluding surfaces exist the supporting bone may offer resistance to break down. In these patients the temporomandibular joint gets most of the trauma resulting from lack of harmony between occlusion of the teeth and the temporomandibular joint in the various excursive movements of the mandible.

That a patient in pain will go to any extremes to get relief is well known to the members of the healing profession. This case is an example of patience and effort by the patient to get relief of pain, also that the temporomandibular joint pathology is a primary dental problem.

PRESENT HISTORY

On May 15, 1951 a patient female, white, age 56, a graduate nurse was referred for periodontal treatment.

Examination of the mouth showed a malocclusion of the anterior teeth, diastemes present between upper anteriors, also spaces between the bicuspids on the right, probably due to loss of right upper first molar. A right upper impacted cuspid had been removed some time ago. Both lower first molars were lost because of caries. The mucous membrane of the mouth in general was of a normal pink color except the marginal gingiva and septal tissue of the right upper and lower molars and bicuspids. This tissue was edematous and hyperplastic in appearance, bleeding very readily.

It was noticed that upon closing the patient seemed to hold the lower jaw in left lateral protrusive position. When the patient was requested to close into complete centric occlusion the lower teeth on right side were all in buccal version. The patient claimed that this position created pain in her right temporomandibular joint.

The patient being employed as R.N. in the Southern Pacific General Hospital, San Francisco, California gave the following history:

PAST HISTORY

In January 1949 she commenced having stabbing pains in the eyeball. These occurred in paroxysms but never entirely disappeared. The pain seemed to have no relation to movements of the eye. There would be periods as long as a week when the pain was not too troublesome. It was always more severe at night, and the eye was hypersensitive to light.

Character of Pain: Sharp, deep, intense over all areas except eyeball. At this point the pain was stabbing accompanied with exquisite tenderness. There were no areas of

Fig. 1. Uncomfortable centric position for patient—note steep lingual inclined plane of disto-buccal cusp causing jaw to move laterally to the left.

superficial hypersensitiveness, no trigger points.

DISTRIBUTION OF PAIN

1. Inner half of eyeball.
2. Inner side of orbital cavity close to nose.
3. Close to and in front of the ear.
4. Supra-orbital over area length of eyebrow.
5. Cheek.
6. All upper and lower teeth.

Examination by the opthomathologist showed no eye pathology. Moderate muscular imbalance was corrected with a change of bifocal lenses. By June 1949 the pain had spread to the area in front of the ear. This pain was distinctly a neuralgia. Dental X-rays were negative. In October 1949, after a complete physical checkup, all tests and examinations including E.N.T. were negative. The combined eye and ear pain continued through the winter of 1949-50. Toothache developed in April 1950. She now had a trio of pain—eye, ear, teeth with less severe pain over the cheek and occasionally some moderately severe pain in the hard palate. Areas of numbness appeared over the skin of the nose and in the tissues of the upper and lower lips.

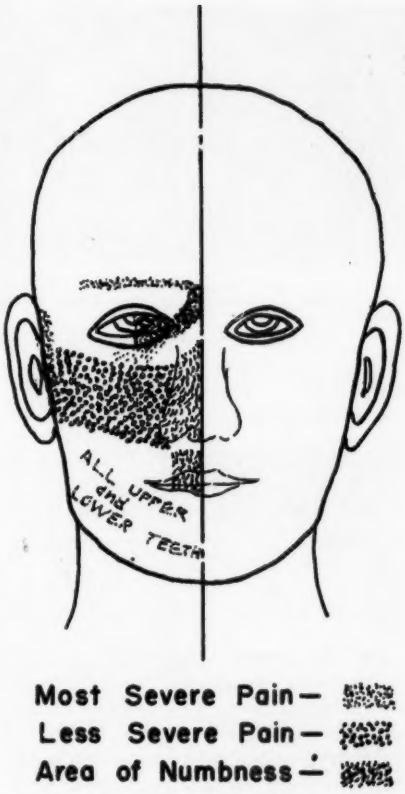

Fig. 1-A. Area of pain distribution.

This true symptomatic facial neuralgia continued to occur in paroxysms at irregular intervals of a few days, and never entirely disappeared. The pain did not seem to be brought on by heat or cold or deep pressure, there were no trigger points, the pain remained sharp, deep, intense. This was diagnosed as Right Trigeminal Neuralgia.

Early in August 1950 while on vacation the eye and ear pain became almost unbearable and was more constant. Another eye, E.N.T. examination gave negative findings. Aspirin gave no relief, neither did codeine. During the last week of the same month she was referred from the E.N.T. clinic to the head of the neurosurgical department

Fig. 2. Comfortable centric position for patient —note space between upper and lower right centrals.

Fig. 4. Left comfortable centric position— note patient has moved jaw to the right bringing all teeth on left side in contact.

for examination and supervision. She was told that there were the following three possibilities:

1. Brain tumor.

2. Aneurysm of the internal carotid artery.

3. Tic Douloureux.

For the purpose of having a complete physical examination she was admitted to the hospital. The neurological examination was negative, there were no signs of increased intra-cranial pressure, the electroencephalogram was negative. Pain was controlled with Demerol 100 mgm. I.M. In October 1950 she was again admitted to the hospital, this time for an angiogram. A perfect picture resulted thus ruling out an aneurysm. Large oral daily doses of vitamin B complex were ordered. The neuralgia was very severe in December, particularly at night.

On January 8, 1951 she discovered that if she moved her mandible to the left, placed her upper and lower teeth in line and held them in that position the pain seemed to gradually decrease in intensity. With this result in mind she found that if she moved the mandible not only to the left but forward so that the upper and lower incisors touched, the pain beginning with that in the eye and supra-orbital area and ending with that in the teeth was gradually and completely relieved. In the above position there was no contact between the upper and lower teeth on the right.

After she had practiced this procedure for several days she reported her findings to the neurosurgeon who confirmed the belief that the neuralgia had been caused by a worn down mandibular process due to an old malocclusion and resulting in pressure on the fifth nerve at the point of articulation.

Fig. 3. Left uncomfortable centric position.

Fig. 5. Anterior view of comfortable centric position. In this position patient was comfortable at all times.

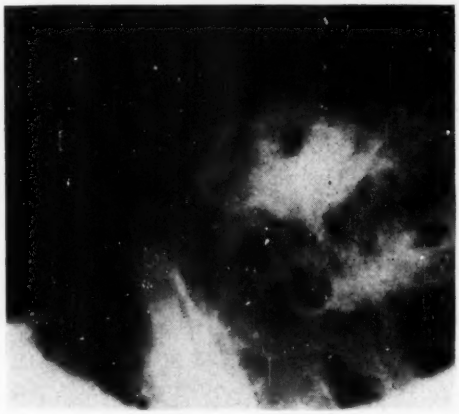

Fig 6. T.M.J. relationship when patient brought teeth into habitual centric occlusion.

Fig. 7. T.M.J. relationship when patient brought teeth into acquired comfortable centric occlusion.

Fig. 8. T.M.J. relationship with mouth completely open.

From January 9th, to February 20th, 1951, except after eating and waking during the night, she was free of pain. A week of pain followed when she carelessly bit on some toast with her right teeth, and in March she had another episode of pain from a similar cause. To avoid the possibility of further return of pain, she decided to avoid all chewing on the right.

With practice, she has become adept in keeping her mandible in the position described. This now requires thought rather than effort. By remembering to do this and to strictly avoid all biting and chewing on the right, she has since been free of pain.

TREATMENT

Temporomandibular joint X-rays were taken to determine the condylar head and fossa relationship when the patient occluded her teeth into what would be considered normal centric occlusion for that case (Fig. 6). T.M.J. X-rays were also taken in what she claimed was a comfortable acquired centric occlusion. The X-rays show that in the acquired position the condylar fossa relationship is more normal while the closed position shows pressure in the right side (Fig. 7, 8).

Scaling and curettage was done. Patient was instructed in proper home care. The edema was reduced on the right side. The occlusion was balanced to permit patient to close into the comfortable position without having to resort to her will power to control the jaw relationship. The patient felt that she had a better resting place for her jaw and more teeth in contact in her acquired comfortable position. Recommendations were made to her dentist for proper prosthetic restorations to make the right side functionable.

SUMMARY

1. This report is made to show to what extent patients will go to get relief.

2. The temporomandibular joint pathology is a medico-dental problem and solution is only possible by joint consultation.

3. The dentist should be more on the alert to recognize the early symptoms and be ready to consult with the periodontist and physician in helping the patient to get relief from pain.

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Gold, Arthur	26 Maple St., Springfield, Mass.
Wellesley Hills	
Young, Arthur Price	169 Washington St., Wellesley Hills, Mass.
MICHIGAN	
Battle Creek	
Uhrie, Ignatz G.	903 Security National Bank Bldg., Battle Creek, Mich.
Detroit	
James, Edgar	1802 David Broderick Tower, Detroit 26, Mich.
Jones, Martha Erma	525 David Whitney Bldg., Detroit 26, Mich.

Grand Rapids
Yeretsky, Willa — 600 Medical Arts Bldg., Grand Rapids 2, Mich.

Maple City P. O.
Gracey, Clayton H. — Glen Lake, Maple City P. O., Mich. (Inactive)

MINNESOTA

Minneapolis
Schaffer, Erwin M. — 1153 Medical Arts Bldg., Minneapolis, Minn.

St. Paul
Johnson, Raymond E. — 824 Lowry Medical Arts Bldg., St. Paul 2, Minn.

MISSOURI

St. Louis
Loeb, Virgil — Beaumont Bldg., 3720 Washington Blvd., St. Louis 8, Mo.

NEBRASKA

Omaha
Donahoe, Lawrence A. — 1124 City National Bank Bldg., Omaha, Neb.

NEW JERSEY

East Orange
Fitzsimmons, Leslie James — Professional Bldg., 144 S. Harrison St., East Orange, N. J.

Ross, Ira — 25 Halsted St., East Orange, N. J.

Jersey City
Schluger, Saul — 30 Journal Square, Jersey City 6, N. J.

Newark
Themann, William A. — 103 Clinton Ave., Newark, N. J.

Weinmann, Isadore — 130 Branford Place, Newark 2, N. J.

Paterson
Hopper, Craft Ackerman — 534 Broadway, Paterson 4, N. J.

Plainfield
Chasens, Abram — 203 W. 9th St., Plainfield, N. J.

Short Hills
Walsh, Joseph Richard — 326 Glen Ave., Short Hills, N. J.

Trenton
Chilton, Neal W. — 225 West State St., Trenton 8, N. J.

Union City
Older, Lester B. — 2115 New York Ave., Union City, N. J.

NEW YORK

Astoria
Drellich, Samuel B. — 2929 23rd Ave., Astoria, N. Y.

Bronx	
Burman, Louis Robert	1749 Grand Concourse, Bronx 53, N. Y.
Goldhaber, Paul	2690 Webb Ave., Bronx, N. Y.
Brooklyn	
Shack, Aaron H.	1 Nevins St., Brooklyn, N. Y.
Buffalo	
Doran, Edward J.	519 Brisbane Bldg., Buffalo 3, N. Y.
Estry, Julius Edwin	333 Linwood Ave., Buffalo, N. Y.
Wolfsohn, Myer D.	175 Linwood Ave., Buffalo 8, N. Y.
New York	
Arvins, Allan N.	4 East 41st St., New York 17, N. Y.
Barkann, Lillian	654 Madison Ave., New York 21, N. Y.
Beube, Frank Edward	730 Fifth Ave., New York 19, N. Y.
Blass, John Lewis	4 East 41st St., New York 17, N. Y.
Golomb, Ida M.	Medical Arts Bldg., 57 West 57th St., New York, N. Y.
Gottsegen, Robert	116 East 53rd St., New York 22, N. Y.
Greenhut, William M.	124 East 84th St., New York 28, N. Y.
Hirschfeld, Isador	730 Fifth Ave., New York 19, N. Y.
Hirschfeld, Leonard S.	730 Fifth Ave., New York 19, N. Y.
Leonard, Harold J.	745 Fifth Ave., New York 22, N. Y.
LeRoy, Louis C.	200 West 59th St., New York 19, N. Y.
McCall, John Oppie	30 West 74th St., New York 23, N. Y.
Merritt, Arthur H.	580 Fifth Ave., New York 19, N. Y.
Miller, Samuel Charles	57 West 57th St., New York 19, N. Y.
Morris, Melvin Lewis	57 West 57th St., New York 19, N. Y.
Roth, Harry	156 East 52nd St., New York 17, N. Y.
Schluger, Saul	116 East 53rd St., New York, N. Y.
Silvers, Herbert	745 Fifth Ave., New York 22, N. Y.
Sorrin, Sidney	745 Fifth Ave., New York 22, N. Y.
Rochester	
Gibbin, Clifford L.	1120 Temple Bldg., Rochester 4, N. Y.

White Plains
Gottesegen, Robert — 175 Main St., White Plains, N. Y.

Yonkers
Witkin, George J. — 45 South Broadway, Yonkers, N. Y.

Chapel Hill — NORTH CAROLINA
Gallagher, J. Wilfred — University of North Carolina, Chapel Hill, N. C.

Charlotte
Mizell, Daniel Britton — 213-16 Professional Bldg., Charlotte 2, N. C.

Walker, Bernard N. — 301 Hawthorne Lane, Charlotte, N. C.

Durham
Kirkland, George F., Jr. — 405 Trust Bldg., Durham, N. C.

Winston-Salem
Watkins, Joseph Conrad — 1507-1511 Reynolds Bldg., Winston-Salem 3, N. C.

Cincinnati — OHIO
Ball, Edward L., Sr. — 3268 Jefferson Ave., Clifton, Cincinnati 20, Ohio

Lytle, Floyd E. — 125 William Howard Taft Road, Cincinnati 19, Ohio

Schott, Carlos H. — #1004 Neave Bldg., Cincinnati, Ohio (Retired)

Cleveland
Biales, Louis — 10465 Carnegie Ave., Cleveland, Ohio

Drake, M. Llewellyn — 1320 Keith Bldg., Cleveland 15, Ohio

Gardner, William M. — 940 Hanna Bldg., Cleveland 15, Ohio

Wiesstien, Leon Martin — 922 Rose Bldg., Cleveland 15, Ohio

Toledo
Robinson, James Robert — Medical Center Bldg., 424 W. Woodruff Ave., Toledo 2, Ohio

Portland — OREGON
Bettman, M. Monte — 528 Medical Arts Bldg., Portland 5, Ore.

Everett, Frank G. — 921 Selling Bldg., Portland 5, Ore.

Lancaster — PENNSYLVANIA
Bentman, J. J. — 155 East Chestnut St., Lancaster, Pa.

Philadelphia
Aiguier, James Edward — 1116 Medical Arts Bldg., Philadelphia 2, Pa.

Alloy, Jack — 1737 Chestnut St., Philadelphia 3, Pa.

Cohen, Abram — 269 S. 19th St., Philadelphia 3, Pa.

Rosenthal, S. Leonard	1424 Medical Arts Bldg., Philadelphia 2, Pa.
Rothner, Jacoby Theodore	1418 Medical Arts Bldg., Philadelphia 2, Pa.
Saturen, Bernard Baer	1930 Chestnut St., Philadelphia 3, Pa.
Stewart, George G.	Medical Arts Bldg., 16th and Walnut Sts., Philadelphia, Pa.
Pittsburgh	
Silverman, Max	5048-49 Jenkins Arcade Bldg., Pittsburgh 22, Pa.
	RHODE ISLAND
Providence	
Lewis, A. Budner	183 Waterman St., Providence 6, R. I.
	SOUTH CAROLINA
Greenwood	
Baker, Stanley Cyril	618-624 Textile Bldg., Greenwood, S. C.
	TENNESSEE
Chattanooga	
Braly, Thomas Edward	1024-27 Volunteer Bldg., Chattanooga 2, Tenn.
Knoxville	
Alford, William Clarence	1816 Clinch Ave., S.W., Knoxville, Tenn.
Memphis	
Friedman, Sidney S.	1003 Madison Ave., Memphis 3, Tenn.
Nashville	
Elam, Roy Oscar	2102 West End Ave., Nashville 5, Tenn.
	TEXAS
Amarillo	
Grupe, Harold Earl	410 Fisk Professional Bldg., Amarillo, Texas
Austin	
Ochsenbein, Clifford	1212A Parkway, Austin, Texas
Dallas	
Collings, C. Kenneth	Baylor University Dental College, Dallas, Texas
Parks, Samuel R.	1505 Medical Arts Bldg., Dallas, Texas
Houston	
Hamblen, Tolar N., Jr.	907 Hermann Professional Bldg., Houston, Texas
Sternlicht, Harold C.	7218 Staffordshire, Houston, Texas
San Antonio	
Nabers, Claude L.	1014 South Texas Bldg., San Antonio, Texas
Waco	
Swindle, John H., Jr.	2505 Washington Ave., Waco, Texas
Wichita Falls	
Nabers, John Moore	401 Hamilton Bldg., Wichita Falls, Texas

Fort Worth	
Prichard, John F.	4121 Camp Bowie Blvd., Fort Worth, Texas
	VIRGINIA
Arlington	
Paulsen, Albert G.	1011 N. Highland St., Arlington 1, Va.
Richmond	
Hart, Virgil Stanley	210 Medical Arts Bldg., Richmond 19, Va.
Lyons, Harry	Medical College of Virginia, Richmond, Va.
Slattern, Richard W.	Medical College of Virginia Dental School, Richmond, Va.
	WASHINGTON
Seattle	
Ingle, John I.	4719 University Way, Seattle 5, Wash.
Thomas, B. O. A.	631 Medical Arts Bldg., Seattle 1, Wash.
	WISCONSIN
Milwaukee	
Sanger, C. N.	3428 W. State St., Milwaukee, Wis.
	ONTARIO, CANADA
Toronto	
Box, Harold K.	86 Bloor St., West, Toronto, Ontario, Canada

U. S. ARMED FORCES AND VETERANS ADMINISTRATION

Belting, Charles M.	V. A. Hospital, 820 S. Damen, Chicago 12, Ill.
Bernier, Joseph L.	Armed Forces Institute of Pathology, 7th St. and Independence Ave., S.W., Washington, D. C.
Brayshaw, Horace Arthur, Lt. Col.	U. S. Army Dental Corps (DC) Dental Clinic, Fort Lewis, Washington
Dummett, Clifton Orrin	Box 516, Veterans Administration Hosp., Tuskegee, Ala.
Moser, Ernest H., Lt. Col.	(DC) USAF 666th Med. Det., APO 20, care P.M., San Francisco, Calif.
Ogle, Merle W., Lt. Col.	(DC) U. S. Army Hospital, APO 43, care P.M., New York City
Scopp, Irwin Walter	Chief, Dental Service, Veterans Administration, New York Regional Office, 252 7th Ave., New York, N. Y.
Toye, Alfred E.	Tokyo Army Hospital, APO 1052, care P.M., San Francisco, Calif.

DECEASED

Cogan, John Vincent	Madison, William James
Cohen, Rose	Marre, Leo Francis
Edwards, Leroy Sherman	Rich, Celia
Hoffer, Carl	Sinclair, J. A.
Hoppe, Walter E.	Smith, Rowe
James, Austin F.	Spalding, Grace Rogers
Kier, George Paul	Tishler, Benjamin
Loftus, J. Leo	Winter, Gordon Richmond

Announcements

SPRING SEMINAR
OF THE
AMERICAN ACADEMY OF PERIODONTOLOGY

The annual Spring Seminar of the American Academy of Periodontology will be held in Ann Arbor, Michigan again this year on May 2, 3, 4. For reservations write Dr. William R. Mann, W. K. Kellogg Foundation Institute, Ann Arbor, Michigan.

Monday, May 2, 1955

9-10 Registration

10-12 "Biology of Virus Disease"
Thomas H. Weller, M.D.
Dept. of Tropical Public Health
Harvard School of Public Health

1:30-4:00 "Oral and Facial Manifestations of Virus Diseases and Their Treatment"
Edward P. Cawley, M.D.
Head of Dept. of Dermatology and Syphilology
University of Virginia

Tuesday, May 3, 1955

9-12 Clinical Laboratory Tests
"Indications and Evaluations of Various Tests Applicable to Dentistry""
Muriel Meyers, M.D.
Associate Professor of Internal Medicine
School of Medicine
University of Michigan
(NOTE) This has not been confirmed.

1:30-3:00 "Electromyographic Changes in Occlusion"
Robert Moyers, D.D.S.
Head of Dept. of Orthodontics
University of Michigan, School of Dentistry

Wednesday, May 4, 1955

9-12 "The Modern Approach to Etiology of Periodontal Disease"
Samuel Charles Miller, D.D.S.
Professor of Periodontia and Chairman of Periodontia Dept.
New York University, College of Dentistry

1:30-4:00 "Operative Dentistry in the Treatment of Periodontal Disease"
Lewis Fox, D.D.S.
Assistant Clinical Professor of Dentistry
Columbia University, School of Dentistry

THE UNIVERSITY OF ILLINOIS COLLEGE OF DENTISTRY

The University of Illinois College of Dentistry will present the fifth of the series of one-day television courses in periodontics on Thursday, February 10, 1955. This is immediately following the Midwinter Meeting of the Chicago Dental Society. As in the past four years, Dr. Balint Orban, Professor and Head of the Department of Periodontics at Loyola University, will direct the course. He will be assisted by Drs. Leo Sreebny, Frank Wentz, Harry Sicher, Harbans Bhatio, and John Kollar.

The complete program will be televised and will include practically every important phase in the treatment of periodontal diseases. It will be of considerable value to the dentist in general practice. The tuition fee is $25.00. For an enrollment blank or further information, write to: University of Illinois College of Dentistry, Postgraduate Studies Extension Division, 808 South Wood Street, Chicago 12, Illinois.

TUFTS COLLEGE DENTAL SCHOOL

The following courses will be offered at Tufts College Dental School during the 1954-55 academic year.

DPG. 601—Special Participation Course in Periodontology. Eight Mondays on the following dates: Nov. 22, 29, 1954-Jan. 3, 10, 17, 24, 31, Feb. 7, 1955. Tuition $200.00. Class limited to 12. Dr. Irving Glickman and Staff.

DPG. 603—Participation Courses in Periodontology. Monday through Friday, April 11-15, 1955. Tuition $150.00. Class limited to 12. Dr. Irving Glickman and Staff.

DPG. 604—Kirkland Gingivectomy Technique. Monday through Friday, May 16-20, 1955. Tuition $125.00. Class limited to 12. Dr. Olin Kirkland.

DPG. 605—Occlusal Equilibration. Monday, Tuesday and Wednesday, Feb. 14, 15, 16, 1955. Tuition $75.00. Class limited to 12. Dr. Edward T. Fischer.

Further information concerning the courses listed above, can be obtained by writing the Director of Graduate and Postgraduate Courses, Tufts College Dental School, 136 Harrison Avenue, Boston, Mass.

SIXTH ANNUAL BERKSHIRE CONFERENCE

Tufts College Dental School announces the SIXTH ANNUAL BERKSHIRE CONFERENCE in Oral Pathology and Periodontology to be held from June 19 to 23, 1955 at Eastover in Lenox, Mass.

The conference consists of four days of lectures, seminars, and panel discussions regarding clinical problems. The faculty includes outstanding teachers and clinicians in dentistry, medicine, and their related fields, who represent teaching institutions throughout the United States and Canada. The conference is arranged so that the mornings and evenings are devoted to professional activities with the afternoons open for recreation.

This conference is for general practitioners as well as individuals with specialized interests. Graduates of dental schools in the United States and other countries may apply. For further information and application write to: Sixth Annual Berkshire Conference, Tufts Dental School, 136 Harrison Ave., Boston, Mass.

BETH ISRAEL HOSPITAL

The Teaching Division of the Dental Department of the Beth Israel Hospital announces a two week conference and seminar in Periodontology from May 9th to the 20th, 1955.

The objective of this seminar is to present to the practitioner all of the techniques employed in periodontal therapy as well as theoretical background. It will include etiology, diagnosis and treatment of various diseases of the supporting tissues of the teeth. Periodontal pocket therapy as well as an over-all mouth rehabilitation program will be stressed. Local environmental factors and systemic influences in periodontal therapy will be discussed. The material will be presented by lectures, demonstrations and clinics. The fee for the course is $350.00.

Co-Directors of Seminar: Henry M. Goldman, D.M.D., Bernard Chaikin, D.M.D.
For further information, write to: Coordinator of Education Programs, Beth Israel Hospital, 330 Brookline Avenue, Boston 15, Mass.

AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES NINTH ANNUAL MEETING

The Ninth Annual Meeting of the American Academy of Dental Medicine will be held at the Park Sheraton Hotel, New York City, on May 13, 14 and 15, 1955.

The scientific program will feature Dr. Herbert S. Kupperman, New York University Bellevue Medical Center, who will discuss the "Stress Syndrome." Dr. Victor Witten of the Skin and Cancer Hospital, New York University Bellevue Medical Center, will present an aspect of dermatology. Drs. Albert L. Stunkard and L. Hinkle, Jr., of Cornell Medical School and New York Hospital, will discuss the "Physiologic and Emotional Aspects of Stress as it Affects the Doctor and the Patient." The field of implant dentures will be covered by Dr. Isaih Lew, who will show movies of patients having this type of restoration.

All members and interested dentists and physicians are cordially invited to attend. Programs and reservations are available through the office of the National Secretary, Dr. William M. Greenhut, 124 East 84 Street, New York 28, N. Y.

"SEMAINE ODONTOLOGIQUE"

The "SEMAINE ODONTOLOGIQUE" (Odontological Week), 62nd Dental Congress, placed under the High Patronage of the President of the French Republic, will be held in Paris from March 31st to April 6th, 1955.

The "SEMAINE ODONTOLOGIQUE" is organized by the National Confederation of Dental Syndicates.

The large Exhibition of Dental Equipment and Products which in 1953 covered an area of 5,000 square meters (54,000 square feet) will have in 1955 a Foreign Section.

For information, apply to: M. Maurice VINCENT, Secrétaire Général de la "Semaine Odontologique," 31, rue Tronchet, PARIS (8e).